

CORONARY THROMBOSIS

A study of 128 cases
with special reference to its relationship to
angina pectoris.

THESIS

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I. INTRODUCTION.

In submitting this thesis it would be proper to begin with an explanation. An extensive material on the subject has already appeared in the medical literature in the past four decades. The usefulness of the study on the general subject at this time might thus be questioned. At the risk of such criticism and with the knowledge that much of the material has already been well described in splendid papers of various capable authors, the Writer all the same considers it worth while to present his observations upon a subject of such vital importance. As heart disease is now the acknowledged greatest single cause of death in the community so coronary thrombosis is the most important cause of heart disease.

It has been my impression that since the end of the war the incidence of coronary thrombosis has been increasing and cases are seen in communities and lands where the condition was almost unknown previously. Furthermore it seems to affect the male population in the prime of life, resulting in considerable loss to the community. This seems true of angina pectoris also. I have attempted to analyse the cases of both coronary

thrombosis and angina pectoris in an attempt to correlate the two conditions or determine any predisposing factors or finally acquire knowledge which may influence the prognosis of these cases.

The thesis is arranged in eighteen Sections.

A historical review is presented in Section II. The incidence of coronary thrombosis as certified cause of death has been investigated by a study of the annual reports of the Registrar General for Scotland and other data and is included in Section III. The case material, Aetiology, contributory and predisposing factors and precipitating factors are dealt with in Sections IV to XIII. Angina pectoris and its relationship to coronary thrombosis is discussed in Section VII . In Section XIV treatment is described and discussed briefly while in Section XV a follow up Study of 50 cases traced is presented.

Finally after presenting Summary and Conclusion in Section XVI the clinical details of 35 cases are given in Section XVII.

Bibliography is attached at the end in Section XVIII.

II. HISTORICAL REVIEW.

The historical development of our knowledge concerning the clinical recognition of coronary thrombosis forms an interesting chapter of internal medicine. Herrick was no doubt the first to draw attention to coronary thrombosis as a condition which does not necessarily cause death but it would be an error to call the symptoms of coronary occlusion ~~as~~ a modern discovery for the medical literature of past century and a half is crowded with clinical and pathological observations upon the condition.

Harvey reported a case which was almost certainly one of coronary thrombosis, although he did not interpret it as such. The interest of it makes it worth quoting. "A noble Knight, Sir Robert Darcy an ancestor of that celebrated physician and most learned man, my own dear friend Dr. Argent, when he had reached to about the middle period of life, made frequent complaint of a certain distressing pain in the chest, especially in the night season, so that dreading at one time Syncope, at another, suffocation in his attacks, he led an unquiet and anxious life. He tried many remedies in vain, having had the advice of almost every medical man. The disease going from

bad to worse, he by and by became cachectic and dropsical and finally grievously distressed, he died in one of his paroxysms. In the body of this gentleman at the inspection of which there were present Dr. Argent then president of the college of Physicians and Dr. George a distinguished theologian and preacher who was pastor of parish, we found the wall of the left ventricle of the heart ruptured, having a rent in it of size sufficient to admit any of my fingers although the wall itself appeared sufficiently thick and strong, this laceration had apparently been caused by an impediment to the passage of the blood from the left ventricle into the arteries".

A number of cases have been reported since Harvey's time and in many of them new signs and symptoms have been observed and emphasized by the authors. The earliest correlation of a high degree of coronary disease with serious illness was made by Bonetus in 1700 in the 2nd edition of his *Sepulchretum* when he described the case of a fat middle-aged poet who succumbed in a few minutes at the onset of "distress in breathing" (which may have been angina pectoris) and who showed at autopsy calcified coronary vessels which were almost if not completely occluded. Huchard (1899) in a pathological analysis of 185

fatal cases of angina pectoris called attention to the importance of coronary artery and to the frequency of coronary thrombosis. In his records all the essential and pathological features of coronary occlusion will be found and he has described the status angiosus, the symptoms of myocardial insufficiency, following the myocardial pain, pericarditis, and the pericardial friction rub, the feeble cardiac impulse and distant heart sounds, the pulmonary oedema, pulmonary infarction, cardiac scars and aneurysm, the sudden death and even recovery from the accident.

Krehl (1901) in his monograph upon diseases of the heart muscles has described clearly the formation of cardiac infarctions and subsequent scar formation and discusses their effect upon the myocardial efficiency. The lack of any constant relation between the extent of scarring and the degree of functional disturbance leads him to surmise that the location of damage is an important factor in determining the occurrence of disorder of function. He comments upon the fact that occlusion of a large branch of coronary artery may go unmarked by any conspicuous symptoms and yet autopsy after years later may reveal an old calcified infarct. In other instances the accident may be accompanied by intense Stenocardia

and the onset of severe myocardial insufficiency. Death may follow instantly or hours or days later or the patient may recover and the gravity of the peril through which he has lived be disclosed only years later when at autopsy a large healed infarct is found. He notes that these scars sometimes give rise to aneurysms which may finally rupture and cause sudden death. Furthermore he discusses the great variation in symptoms following coronary occlusion and points out that the symptoms are likely to be mild when there is diffuse Sclerosis of the vessels and that they are likely to be severe when the vessels are relatively healthy and the occlusion occurs suddenly. Finally he confesses to mystification at the remarkable suddenness with which death often comes. This is not the death of circulatory failure. He is convinced that under these circumstances something extraordinary goes on in the heart which has not been satisfactorily explained.

Coronary thrombosis (occlusion) has been considered an episode in the progress of angina pectoris, a harrowing episode it is true and yet but the dramatic ending of an interesting and varied clinical story. The history of angina pectoris is associated with the names of celebrated men like Heberden, Parry, Jenner and Hunter.

On July 1768, Heberden read his paper entitled "Some account of a disorder of the breast", in which he stated that, "there is a disorder of the breast, marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it and not extremely rare of which I do not recollect any mention among medical authors. The seat of it and sense of strangling and anxiety with which it is attended may make it not improperly be called Angina pectoris". By using the word angina (strangling) it is clear that Heberden differentiated angina pectoris from multiple forms of chest pains of varied etiology, which were loosely assembled under the heading of dolor pectoris. Heberden was the first to describe angina pectoris, a symptom complex, due to disorder of the heart, but he did not recognise that the disease of the coronary arteries was the primary factor. It was however left to Jenner and Parry (1799) who observed that calcification of the coronary arteries was the real lesion.

In 1773, John Hunter had his first attack of angina pectoris, which was narrated by his nephew Edward Home. In 1776 he had another attack and when recovering from it he visited Bath, where he was seen by Edward Jenner, who wrote to Heberden, giving his diagnosis of John Hunter's case. He

suggested for the first time the probable association of disease of coronary arteries with angina pectoris. In 1793 an autopsy of John Hunter's body revealed the coronary arteries to be converted into open bony tubes". This decided advance in the pathology of angina pectoris was communicated by Jenner to Parry who brought it before the medical profession. Jenner and Parry (1799) showed that angina pectoris was associated in most cases with an ossification or some other form of obstruction by disease of the coronary arteries. In spite of this pathological finding William Stokes (1854) discussing angina pectoris and sudden death, states that "we may conclude that in the special group of symptoms described by Heberden and Parry, obstruction of the coronary arteries may or may not be present and as a cause of angina, its action is remote and its existence unnecessary".

Jenner's communication to Heberden and Parry's publication of Jenner's work appears to have been ignored by the majority of the physicians of the nineteenth century as the cases of sudden death following severe anginal pain, revealing at autopsy gross disease of the coronary occlusion were attributed to fatty degeneration of the heart, acute myocarditis, rupture of heart or to aneurysm

of the heart, while angina pectoris, although showing frank evidence of disease, was described as "neuralgia of the heart".

Hope (1831) discussing death in cases of "Softening of the heart" states that there are two varieties of softening:- (1) Softening with increased intensity of redness - namely, claret, morone, or violet coloured denoting an excess of blood in the muscular substance and (2) Softening with diminution of redness, namely faint yellow or fawn coloured and bespeaking a deficiency of blood". Laennec supposes softening of the heart to be "an affection sui generis, resulting from a derangement of nutrition", while Bouillaud contends that Softening in all its varieties of colour is a result of inflammation. Coronary arteries have not been mentioned by these two writers in their papers. Hope states that neuralgia of the heart or angina pectoris may originate from any cause, whether organic or functional, capable of irritating the heart or of rendering it morbidly susceptible to irritation, and as a structural disease of the organ has this effect more than any other cause, it is that on which the malady is most frequently dependent." The most violent cases of angina seen by Hope "have been connected with osseous, cartilaginous, steatomatous or other

degeneration of the heart or great vessels and especially of the coronary arteries.

Stokes '1854) states that fatty degeneration often causes sudden death, the obstruction of the coronary arteries is often met with in this disease and that in certain cases it may assist in the production of atrophy and the fatty state of heart, "but that we may often look on this condition as one of the adjuncts rather than the primary cause of the disease". In the chapter on angina pectoris he says that whatever may be the immediate cause of the pain and sensation of approaching death these are often encountered in connection with some form of organic disease of the heart, namely:- (1) weakness and attenuation. (2) weakness with fatty degeneration. (3) some form of valvular disease generally affecting the left side. (4) Disease of the aorta with or without obstruction of the coronary arteries.

A clear description of what almost appears to be a case of coronary thrombosis is Latham's (1876) account of the death in 1842 of the celebrated Dr. Arnold of Rugby at the age of 47 years. Latham referred to the case as one of spasm of the heart. He mentions environmental factors and heredity as playing a part in the aetiology of the condition. Latham's description of the case is as follows:- "On my entering Dr.

Arnold's room, he said he was sorry to disturb me so soon, and that he had not sent for me before, thinking that it would go off". He added, "I have had very severe pain since 5 o'clock, at intervals, and it gets worse. I think the pain was seated at the upper part of the chest towards the left side and extended down the left arm". He had been rather sick. His pulse I could scarcely feel. The tongue was clean. There was cold perspiration over his face. He then asked me what the pain was? I told him I believed it to be spasm of the heart. I asked him whether he had ever fainted in his life. No, never. If he had at any time difficulty of breathing. No, never. I then asked him if any of his family had ever any disease of the chest. "Yes, my father had, he died of it." He enquired if disease of the heart was fatal. I answered that it was. "Was it a common disease". I said "not very common". "Where do you find it"? "In large towns, I think". "Why"? Perhaps from anxiety and eager competition among the higher and intemperance among the lower classes. Dr. Arnold died a little more than half an hour after I first saw him. The postmortem examination revealed a soft flaccid heart muscle. There was but one coronary artery that considering the size of the heart was of small dimensions. It

presented a slight atheromatous deposit an inch from its orifice".

The association of coronary vessels disease and anginal pain as described so well by Latham was again lost sight of by other investigators although heredity and higher grades of society were recognised as contributory factors in cases of sudden death.

Schrotter (1878) described ~~Spasm~~ Spontaneous rupture of the heart and acute myocarditis. His description was almost that of a myocardial infarction as a result of coronary thrombosis. Regarding Spontaneous rupture of the heart Schrotter quotes Moragagani who wrote at length concerning it and gave expression to the opinion that it could only occur where muscle tissue was diseased. Schrotter states that disease of the coronary arteries is not infrequently a cause of rupture. As regards sex, it is very much more common among men than women. Discussing the symptoms he states that often death occurs so quickly that the patient does not utter a sound or sometimes gives a short cry. They fall to the ground, pallid, breaking out in a cold sweat with a pulse gradually failing in power, soon lose consciousness and after a few slight convulsions die. Again on the other hand many cases live for

hours and days and the symptoms may begin with pain or a feeling of oppression in the chest, causing great anxiety, the pain extending across the breast and down the left arm. Such attacks as these may be repeated with more and more frequency, until finally in one of them the patient suddenly passes away. Schrotter also stated that the first evidence of acute myocarditis may be a rupture of the heart or an attack of apoplexy (embolic). A man previously in good health begins to complain of great weakness and lassitude, of pain in the region of the heart or of a feeling of oppression, which may suddenly increase to the greatest agony. Soon with this are associated attacks of Dyspnoea, irregular action of heart, fainting etc. From Schrotter's pathological description^{of}/these lesions there can be little doubt that these correspond to infarction of the myocardium, yet no definite mention was made of the coronary arteries in his discussion.

It was to Sir William Osler (1897) that we are indebted for the first real correlation of the clinical and pathological findings. In his study of the material which came from his private practice he gave a very thorough discussion of practically every aspect of angina pectoris, though many of his cases termed angina pectoris

would now be called coronary thrombosis. He recognised the definite relationship between sudden death and disease of the coronary arteries and angina pectoris. Regarding the predisposing causes he says that men of muscular, even athletic, build who have been devotees of Bacchus and Venus form the largest contingent, while more wise men than fools are the victims of this disease. He emphasizes the heredity factor and states that true angina pectoris is an arterial incident and since members of certain families show a special tendency to arterial degeneration, it is not surprising to find cases in father and son, or in brothers or even in representations of three generations. A very good example of which is the Arnold family:- William Arnold died suddenly of spasm of the heart in 1801, his son celebrated Thomas Arnold died in his first attack in 1842 aged 47. Matthew Arnold, the latter's distinguished son was also a victim and died suddenly in 1888 aged 66 years.

From the above review it is evident that what are now recognized as cases of acute coronary thrombosis were generally regarded as incidents of severe angina pectoris or Status angiosus. It is this distinction between attacks of angina pectoris and attacks of coronary thrombosis which has

developed since 1910 i.e. four decades ago.

Dock (1896) was one of the very first to report a case of coronary thrombosis diagnosed ante mortem and proved at autopsy. One of his patients, an elderly man, had felt breathless on exertion for three months. Seven days prior to death he had a severe pain in the cardiac region and fainted, becoming pulseless and very dyspnoeic. Evidence of left ventricular failure developed. Pericardial friction rub was heard at the apex. Death occurred suddenly while he was straining at stool. Post mortem revealed severe arteriosclerosis of coronary arteries and occlusion of the anterior descending branch of the left artery and a branch of the circumflex artery by a thrombus. The myocardium was infarcted with an apical pericarditis. Dock states that in this case the relation of coronary sclerosis to the gradually developing dyspnoea and the infarction to the acute attack a week before death is clear. A heart extensively necrosed may continue to act for some time fairly well if not exposed to abrupt strain, thus explain the early stages of these cases in which large fibrosis is found post-mortem in the heart. It is also a fine example of atheroma limited almost entirely to the coronary vessels. Dock recognised the importance of the

pericardial friction rub as an aid in diagnosis of coronary thrombosis.

The first satisfactory account of the clinical features of an attack of coronary thrombosis was published by Obratzow and Strascheko (1910). These Russian authors diagnosed correctly two of the three cases they published. They emphasized a triad symptom:- Severe lasting retro-sternal pain, dyspnoea and orthropnoea and finally gastralgia. They called attention to many of the features now recognised as important findings, such as Gallop rhythm, Cheyne-Stokes breathing, pericardial friction rub, distant heart sounds, mural thrombi, pale cyanosis etc. The fever present in one of their cases, they believed was due to pericarditis and pleuritis. Hocchaus (1911) similarly reported four cases of which two were diagnosed antemortem.

Herrick's careful observations and persistent effort really focussed the attention of the American medical profession on this disease and in 1912 he emphasized the fact that coronary thrombosis was a clinical entity, that could be recognized during life and it need not end fatally. Curiously enough this publication did not produce the desired effect for it aroused no interest. After a lapse of six years (1918, published 1919)

he enlarged on his original observations and on this occasion had a more receptive audience. In the same year Levine and Tranter (1918) published a report of two cases of coronary thrombosis quite independently of Herrick as at that time they were unaware of his previous work. One of their cases was diagnosed antemortem. Libman was also aware of the condition as differing from ordinary attacks of angina pectoris. In 1916 while discussing the different kinds of chest pains he mentioned in passing, "the diagnosis of a recent thrombosis could often be facilitated by the development of a slight temperature, moderate leucocytosis and evidence of a patch of pericarditis all coming on within a couple of days after the attack of a severe pain". Levine and Tranter (1918) also observed fever and leucocytosis in coronary thrombosis and they pointed out that this association of fever and leucocytosis in epigastric pain together with other features might strongly simulate an acute surgical abdominal condition. The first of their two cases was operated on by mistake as an acute surgical emergency and the second was saved from this error and diagnosed antemortem by their previous experience.

Shortly after these publications an extensive literature appeared and amongst the more important

articles of clinical nature were those of Gorham, Paullin, Levine, Longcope, Thayer, Wearn, Hamman, Benson and Wolf and White. These various publications served to bring the clinical features of coronary thrombosis before the medical public in the United States so much so that Christian (1925) remarked, "cardiac infarction stands out as a clean cut entity, easy of recognition by any physician who knows the symptoms and usual findings". Some of the above authors emphasized particular points that lead the way for a clearer understanding of the subject. Gorham (1920) pointed out the diagnostic importance and frequency of a pericardial friction rub, while Wearn (1923) beside giving a very clear description of the symptoms and signs of the disease called attention to a marked diminution in the height of the waves in the electro-cardiograms in a few of his cases. Levine (1925) brought out the interesting observation that those patients who had hypertension and angina pectoris before the attack of coronary thrombosis might become free from anginal attacks if recovery was attended by a permanent and distinct lowering of the blood pressure.

While this extensive literature was appearing in America, it is surprising that it was not until 1925 that this subject of coronary thrombosis as a

specific problem began to be considered in United Kingdom. Allbutt (1915) had however discussed coronary thrombosis chiefly as it affected and opposed his view of the aortic origin of angina pectoris. Mackenzie (1923) though not making the clinical distinction from angina describes typical examples of coronary thrombosis.

McNee (1925) reported three cases of coronary thrombosis and stated that in sudden thrombosis of large branches of coronary arteries there may occur in patients who survive immediate shock a very remarkable clinical syndrome which deserves attention. The main clinical features of the syndrome are:-

- (a) An agonising pain of varying distribution, which lasts longer than the usual attack of angina pectoris.
 - (b) Dyspnoea which may be extreme.
 - (c) A peculiar colour and appearance of the face.
 - (d) Immediate signs of acute cardiac failure.
 - (e) One sign which is inconsistent but almost pathognomonic in association with a suggestive history or group of symptoms is a localised pericardial friction rub.
 - (f) Fever and polymorphonuclear leucocytosis.
 - (g) Various abnormalities in the electrocardiogram.
- He further stated that the recognition of this clinical syndrome should be fairly easy in patients who have suffered from

cardiac complaints such as angina pectoris. The real difficulty in diagnosis arise when coronary occlusion is the first evidence of cardiac disease in a previously healthy patient. The publications of Gibson (1925) and Parkinson and Bedford (1928) revealed that they were also aware of this ailment. Campbell (1929) emphasized the great frequency of coronary vessel disease in the general population. The majority of his autopsy cases were those following surgical operation for widely different diseases, the coronary arteries in these cases revealed a greater or lesser degree of lesion.

A most important and helpful advance in the clinical recognition of coronary thrombosis came about when certain Electro-cardiographic changes were found to be fairly characteristic of the acute stage of this condition. This work was some experimental observation in dogs by Smith (1920). He noted sharp inversion of the T wave of the electrocardiogram in dogs.

The discovery of Pardee (1920) that during the early days following an attack of coronary thrombosis, there are fairly characteristic changes in the electrocardiogram has proved to be a most valuable addition to diagnostic methods. These changes were described of consisting of a high take off the T wave from the descending limb of the

R wave. This alteration does away with the customary iso-electric interval that normally is found between the R and T waves. It was also noted that T wave goes through rapid changes during subsequent days after the attack and may become sharply inverted. This publication was soon followed by others which confirmed and somewhat elaborated the cardiographic evidence of coronary artery disease. These authors found the characteristic changes in R-T. interval as soon as six and a half hours after the onset of the attack.

A recognition of the classification of heart disease to include coronary thrombosis in the "International list of causes of death occurred in 1930. Prior to 1930 there was no separate classification of diseases of the coronary arteries though earlier literature contained many references to coronary vessels in relation both to angina pectoris and to cases of sudden death. It is not therefore surprising that cases of sudden death due to coronary occlusion were termed, "acute myocarditis", "fatty heart", "Spontaneous rupture of the heart" or else the all embracing term "Angina pectoris".

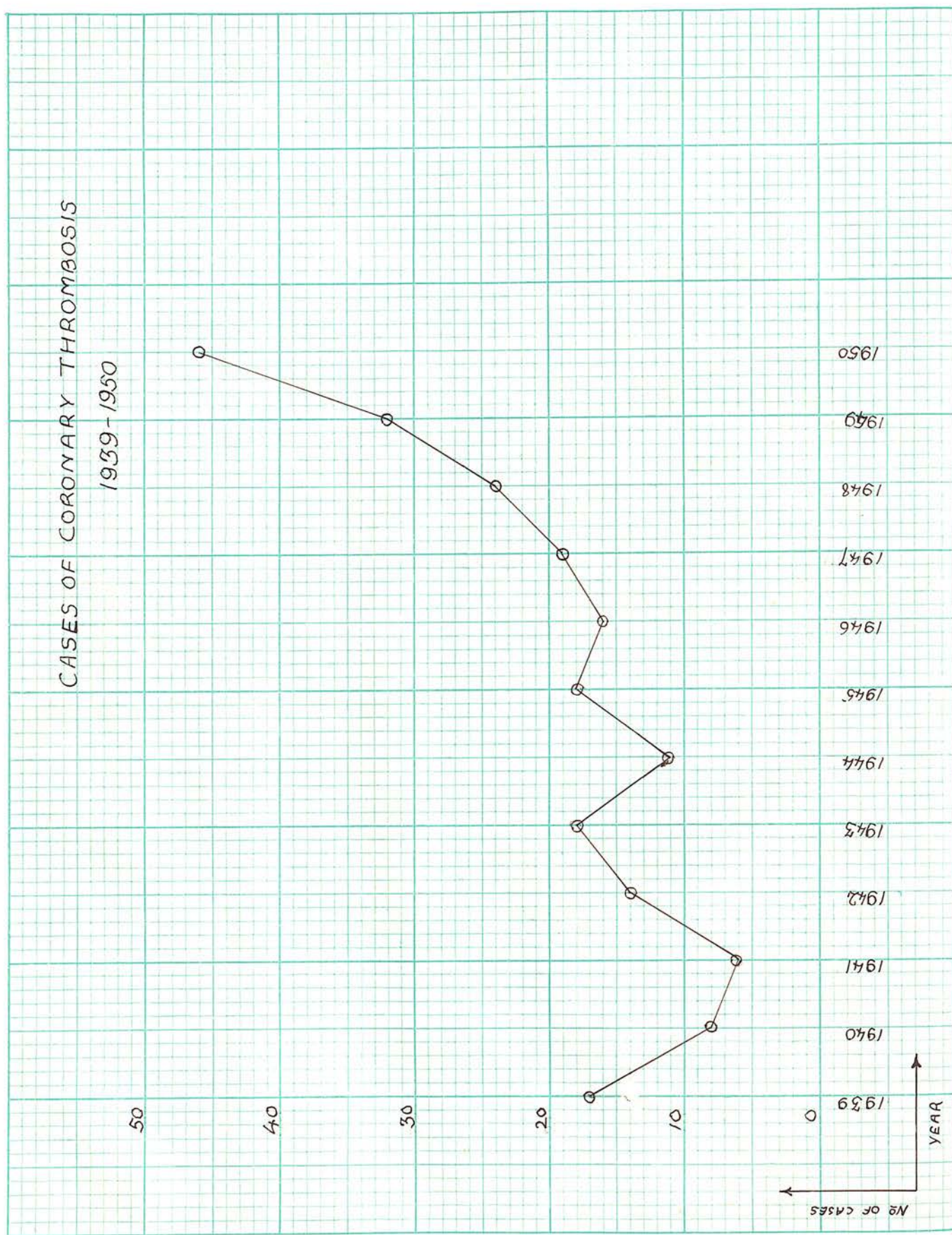


Fig. I

CASES OF CORONARY THROMBOSIS AND TOTAL NO OF CASES ADMITTED 1939-1960

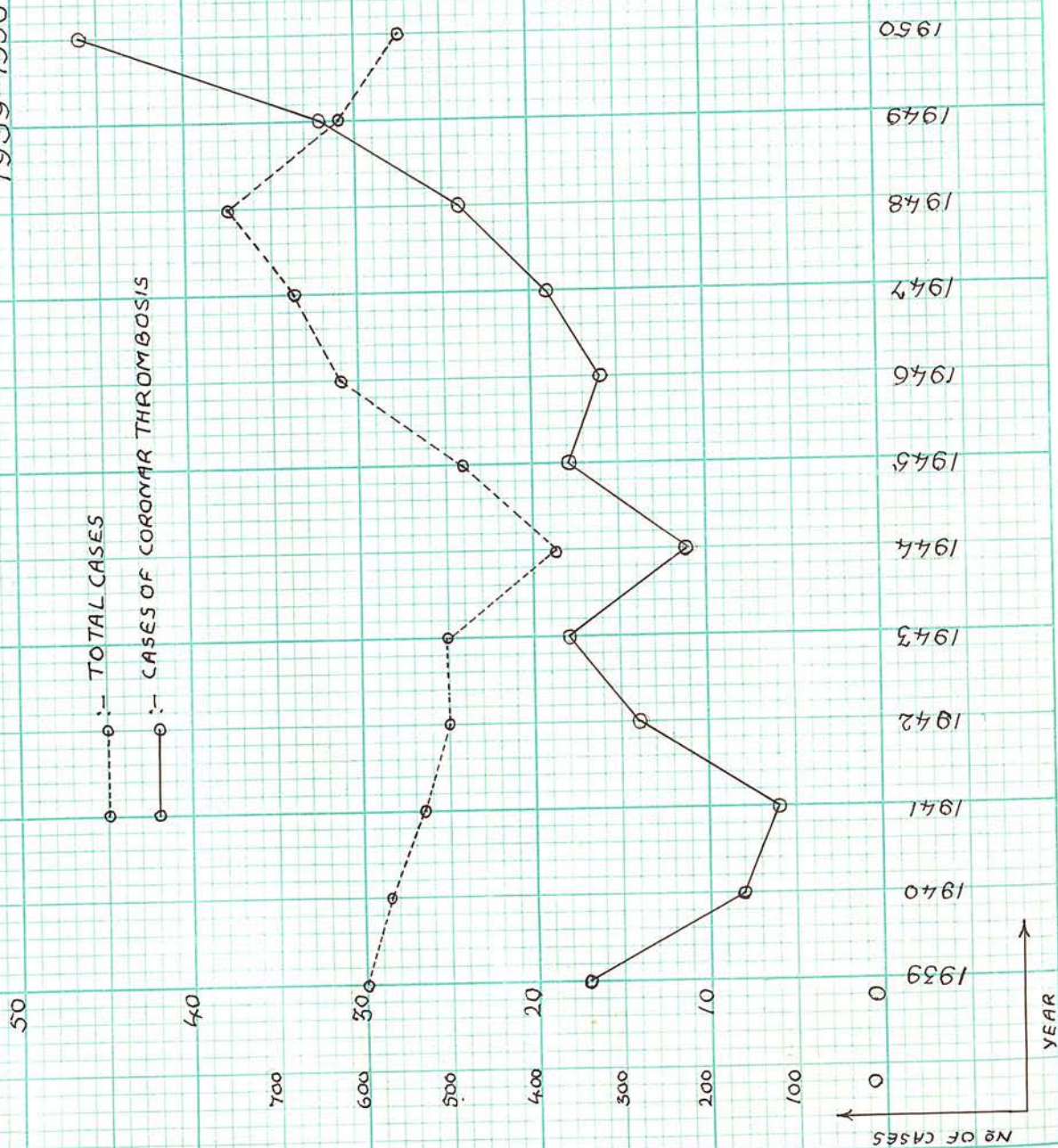


Fig. II

III.

THE INCREASED INCIDENCE OF CORONARY THROMBOSIS.

Coronary artery disease has increased in the past decade or two and it is now the common type of heart ailment seen in a hospital ward. This is especially true of coronary thrombosis with myocardial infarction and is due partly to decreased death rate from infectious diseases in early life, the greater percentage of the population thus reaching the age groups commonly affected with atherosclerotic diseases. Advanced medical knowledge, improved diagnostic technique and the recognition of a distinct clinical picture of the disease have also to some extent been responsible for this increase. Table I shows the incidence in a hospital ward during the last decade.

Table I.*

The cases of coronary thrombosis admitted to the wards of Professor D.M. Lyon in R.I.E. from 1939 to 1950.

Year	Total No. of cases admitted	Cases of coronary thrombosis
1939	604	17
1940	570	8
1941	531	6
1942	501	14
1943	505	18
1944	372	11
1945	480	18
1946	619	16
1947	675	19
1948	749	24
1949	624	32
1950	558	46

* Fig:- I, II.

These figures show that there has been a steady increase in cases of coronary thrombosis from 1939.

Friedberg writes that in the United States the cardiac mortality in 1940 was 292.5 per 100,000 population and 25 per cent of this was attributed to diseases of coronary arteries. Most of the latter undoubtedly represented coronary thrombosis and its consequences. There has been a progressive increase in the reported, if not actual incidence, of coronary thrombosis. In America the crude death rate from coronary disease had arisen from 7.9 per 100,000 in 1930 to 23.1 in 1935. Subsequently this increased to 71.4 in 1940 and 95.2 in 1946. Denny (1936) in a study of deaths from heart disease in Brooklyn from 1900-1935 observed that in the period 1900-1904 the word coronary did not appear on a single death certificate. From 1905 to 1924 the rate for coronary disease increased from 3.9 to 13.9 per 100,000 of the population. 1930 to 1936 the increase was very rapid, the rate being 58.7, 76.6, 94.1, 101.2 and 140; and in 1935, 156.6. This author considered that a part of the increase was due to the more frequent diagnosis of coronary thrombosis than before. In Philadelphia, Hedley (1939) noted an increase of 126 per cent in the

reported mortality from acute coronary occlusion in five years. This study was based on 5116 deaths reported to be due to acute coronary thrombosis during the period from January 1st, 1933 to December 31st 1937. Master (1947) estimated that 800,000 attacks of coronary thrombosis occur annually in the United States and using the United States census figure for the number of men and women over 40 years of age, it meant that approximately 1 man in 40 and 1 woman in 115 suffer from coronary thrombosis yearly. Master attributed the increase in incidence of the disease to (1) the lengthened span of life, (2) ageing of the population, (3) improved diagnosis and treatment, and, (4) accuracy in terminology. Cassidy (1946) was of the opinion that the increasing prevalence of coronary disease was beyond question. He reported that in the year 1926, 64,645 persons died in the United Kingdom of all forms of heart disease. In 1936 this number was 126,584. The figures for coronary disease were even more startling. 1880 died in 1926, 14,095 in 1936 and 19,496 in 1939. Part of this rapidly increasing death rate from coronary disease was no doubt attributable to the increasing age of the population, as in 1900 there were 1,750,000 persons over 65 years of age in Great Britain and in 1937 there

were over 3,750,000 (Dudley Committee 1944). There had also been an increase in the certification of deaths from coronary thrombosis since McNee (1925) brought to the notice of the physician in this country the clinical features of coronary thrombosis. Even so it was his (Cassidy's) impression that coronary thrombosis was far more prevalent than it had been.

Ryle and Russell (1949) have made a study of both angina pectoris and coronary thrombosis. These authors have analysed the reports of the Registrar General for England and Wales during the period from 1921 to 1945. The statistical pattern for both sexes is similar. Each five-year age group between the ages of 40 and 75 years revealed an increase of approximately 250 per cent in the mortality due to the coronary artery disease in 1931-39 as compared with 1921-30. The increase continued and in 1941-45 it ultimately amounted to approximately 450 per cent. In middle age, 40-55 years, the male death rate had increased more rapidly than that of females, whereas in old age the position was reversed. In 1940-45 the male death rate between 40 and 50 was more than five times greater than the female. At 55-60 the ratio dropped to 4.0 and at 75 it was less than 2. Considering (a) the changing fashion in diagnosis,

(b) changes in the international classification of causes of death, and (c) the abandonment by the Registrar General of a priority classification and his acceptance of the sequence as stated by the doctor when more than one cause of death is mentioned on the death certificate, Ryle and Russell concluded that there was an increase in death due to coronary disease. Although the increase was definite they considered that it was now less rapid than in the period before the war 1939-45.

It is reported in the 95th Annual Report of the Registrar General for Scotland that in 1949 in Scotland deaths from heart disease numbered 19,347 and is the largest number ever recorded. This number increased steadily year after year with scarcely a break from 9,535 in 1931 to 16,503 in 1940. Change of classification reduced this number only by about 1 per cent and the following two years showed a considerable reduction, but the number increased steadily to 18,537 in 1947. It dropped again in 1948 to 17,566. The number of deaths from diseases of coronary arteries and angina pectoris was 5,994 compared with 5,354 in 1948. These deaths which in 1931 numbered only 823 have shown an increase in every year since. There is no doubt, however, about the marked increase in deaths from coronary thrombosis and angina pectoris.

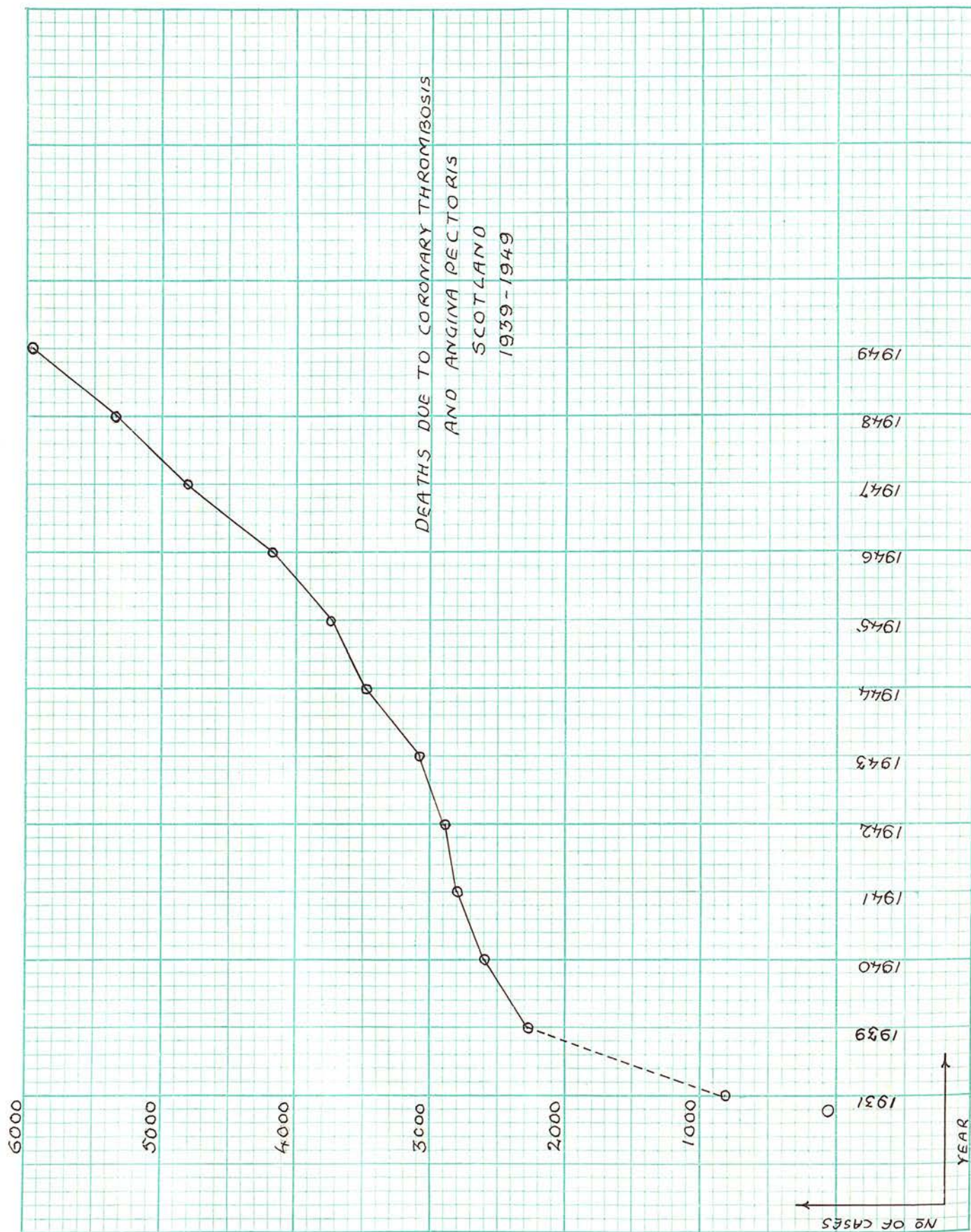


Fig III

DEATHS DUE TO CORONARY THROMBOSIS AND ANGINA PECTORIS SCOTLAND 1939-1949

MALE
FEMALE

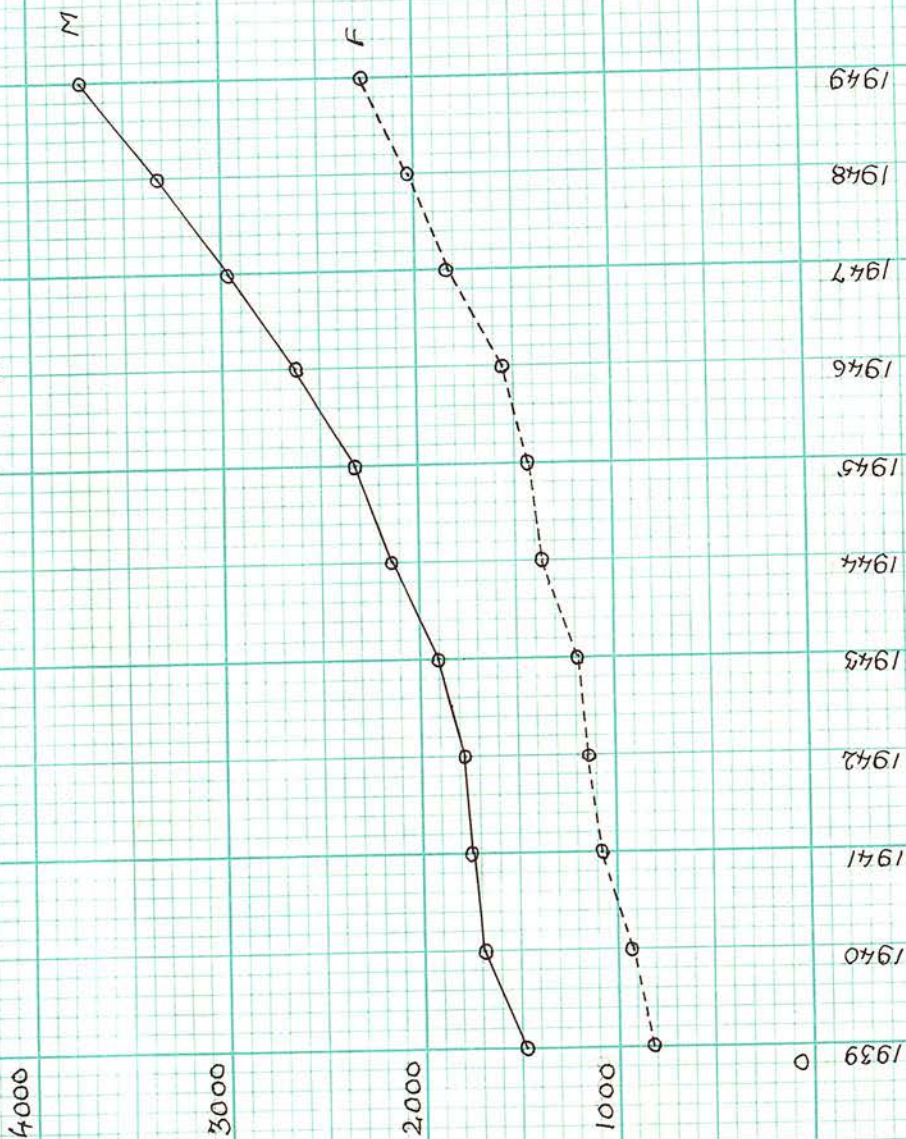


FIG 7V

For example in 1941 there were 184 deaths from angina pectoris and 2619 from coronary thrombosis and embolism while in 1949 the number of deaths from under the combined heading is 5,994. Male deaths from this cause numbered 3,740 as against 2,254 among females.

Table II. *

Comparison in deaths due to disease of the coronary arteries and angina pectoris in Scotland from 1939 to 1949:-

<u>Year</u>	<u>Males</u>		<u>Females</u>	
1939	1482		811	
1940	1688		925	
1941	1624	angina 124	995	angina 60
1942	1672	" 89	1083	" 34
1943	1833	" 69	1144	" 25
1944	2109	" 23	1343	" 14
1945	2277	" 35	1395	" 9
1946	2635		1550	
1947	2989		1834	
1948	3320		2034	
1949	3740		2254	

Vital Statistics and data from other sources call attention to the considerable increase in heart disease. Figures of the Registrar General for Scotland gave the number of deaths from heart disease, 9,535 in 1931, and 16,503 in 1940 and it steadily increased to 18,537 in 1947 while in 1949 the number was 19,437, the largest number ever recorded. Thus the march of time witnesses the increasing slaughter of humanity by heart disease and in the vanguard of this malicious host is

* Fig:- III, IV.

coronary disease which no longer respects certain age groups and is progressively depleting the ranks of younger persons.

There is no doubt about the increase in the incidence of coronary thrombosis and angina pectoris. In Scotland in 1941, there were 184 deaths from angina pectoris and 2,619 from coronary thrombosis, while in 1949 the number of deaths from combined cause rose to 5,994. One of the disconcerting facts regarding the present situation is the increasing incidence of coronary thrombosis among younger persons. It is now common in the 5th decade of life, appearing more frequently among those in the 4th decade and occasionally is observed among those in the third.

The increase in coronary disease is attributed to worry and to the stress and strain of modern life - that is nervous and emotional strain. Attention has also been called to the relative freedom from coronary disease of those doing manual work. The explanation may be that those who suffer least have occupations, which entail a certain amount of daily physical activity, while those who suffer most have sedentary occupation which requires a minimum of physical activity. It may be that in those with sedentary occupations, nervous and emotional strain be a contributing

factor and sedentary life, the insufficient muscular activity a possible basic underlying cause.

IV. CASE MATERIAL.

The present study consists of 128 cases of coronary thrombosis treated in the wards of Professor D.M. Lyon, Royal Infirmary, Edinburgh, from 1st January 1948 to December, 1951. These were diagnosed (1) clinically, (2) electrocardiographically, and, (3) by demonstration of recent myocardial infarction at autopsy. These cases occurred in the general population of a busy industrial district where many people live under the "stress and strain of modern life". These figures may well be different from those in rural districts.

V. AETIOLOGY OF CORONARY THROMBOSIS.

Coronary thrombosis refers to the disease associated with an acute thrombotic occlusion of a major coronary artery. The thrombosis is almost always a complication of coronary arterio-sclerosis. Acute coronary occlusion is a more inclusive term denoting the sudden obstruction of a coronary artery, either by the development of a thrombus or by an intimal haemorrhage with swelling of the arterial wall or by embolus.

The underlying causes of this condition are:-

1. Arterio-sclerosis of the coronary arteries which is responsible for at least 95 per cent of the cases of acute coronary occlusion.
2. Syphilitic aortitis which is commonly complicated by Stenosis or occlusion of the coronary ostia.
3. Coronary embolism is the commonest of the rare causes of acute coronary occlusion.
4. Periarteritis nodosa.
5. Rheumatic fever has rarely been associated with acute coronary occlusion and myocardial infarction in children.
6. Thrombo-angitis obliterans.

The commonest cause of myocardial infarction is the narrowing of coronary arteries due to

atherosclerosis with a superadded thrombosis.

The contributory and predisposing factors in the aetiology of this disease are many and varied and will be dealt with in this study.

VI. CONTRIBUTORY AND PREDISPOSING FACTORS.

SEX INCIDENCE.

In the group of 128 patients with acute manifestation of coronary thrombosis there were 97 males and 31 females, thus giving a sex ratio of 3 males to 1 female. This is the usually accepted figure. There is a notable preponderance of males among patients suffering from acute coronary thrombosis.

In the series of Bellings et al (1949) from Nashvill the sex ratio was 2.7 males to 1 female. Chambers (1946) in New York had a ratio of 3 males to 1 female, Mintz and Katz (1947) in Chicago gave figure of 2.2 males to 1 female. Levine and Brown (1929), Parkinson and Bedford (1928), Conner and Holt (1930), Willius (1936), Bean (1937), Master et al (1939) gave ratios of 3.26 males to 1 female, 13.3 males to 1 female, 5.5 males to 1 female, 7 males to 1 female, 2.2 males to 1 female, 3.4 males to 1 female, respectively in their series of cases.

The sex ratio varies from 2.2 to 1 reported by Katz and Mintz (1947) and Bean (1937) to 13.3 to 1 of Parkinson and Bedford (1928).

Table III.

Sex incidence of coronary thrombosis reported by various authors.

<u>Name of Authors</u>	<u>Male</u>	<u>Female</u>	<u>Number of cases</u>
1. Parkinson and Bedford (1928)	13.3	1	100
2. Levine and Brown (1929)	3.26	1	145
3. Conner and Holt (1930)	5.5	1	287
4. Willius (1936)	7	1	370
5. Bean (1937)	2.2	1	300
6. Master et al (1939)	3.4	1	500
7. Rathe (1942)	2.4	1	274
8. Cassidy (1946)	3.5	1	1000
9. Chambers (1946)	3	1	100
10. Fisher and Zukerman (1946)	2.72	1	108
11. Katz and Mintz (1947)	2.2	1	572
12. Bellings et al (1949)	2.7	1	240
13. Present Series	3.129	1	128.

The ratio of 3 to 1 based on studies of cases at autopsy is probably more accurate than the higher ratios reported in the clinical series.

The males are more prone to develop coronary thrombosis than females. No explanation has been given for this increased incidence in males. Hypertension, occupation and smoking may be possible factors. It has been suggested as a hypothesis, however, that as a result of woman's child bearing function, nature has given her a cholesterol clearing mechanism which may be an important protective factor against the development of coronary disease. Fellner, in reporting his findings in autopsies on atherosclerotic subjects, observed that as a rule women develop arteriosclerosis later in life than men and that they also suffered a milder form of the disease. Dock (1946) found that the intima of the coronary arteries lying in the epicardium, already known to

be thicker than that of any artery of similar calibre elsewhere in the body, is much thicker in males than in females. This is believed to establish the basis for the sex difference in the incidence of coronary occlusion and to prove that the predilection of atherosclerosis for the coronary arteries is due to their possessing an intima varying in thickness from 10 to 600 per cent of that of the media and averaging 26 per cent in newborn males and 8 per cent in females. It may be that those born with a greater degree of thick endothelium develop more atheromatous changes in these areas. Another possible explanation is the peculiarities in the structure and function of the endocrine system which may be the basis of premature arteriosclerosis.

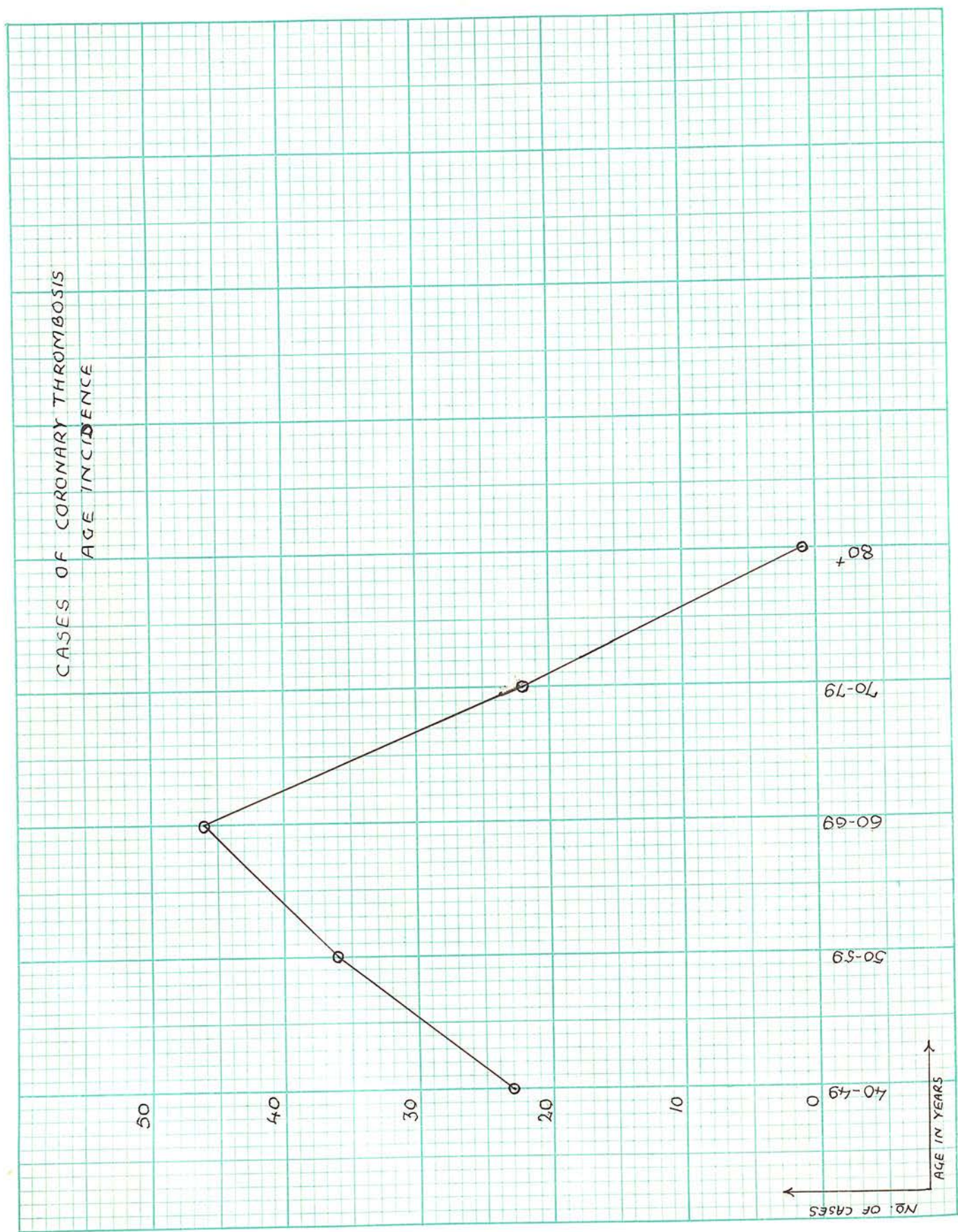


FIG V

CASES OF CORONARY THROMBOSIS

AGE DISTRIBUTION

MALE

FEMALE

○

○

○

○

50

40

30

20

10

40-49

50-59

60-69

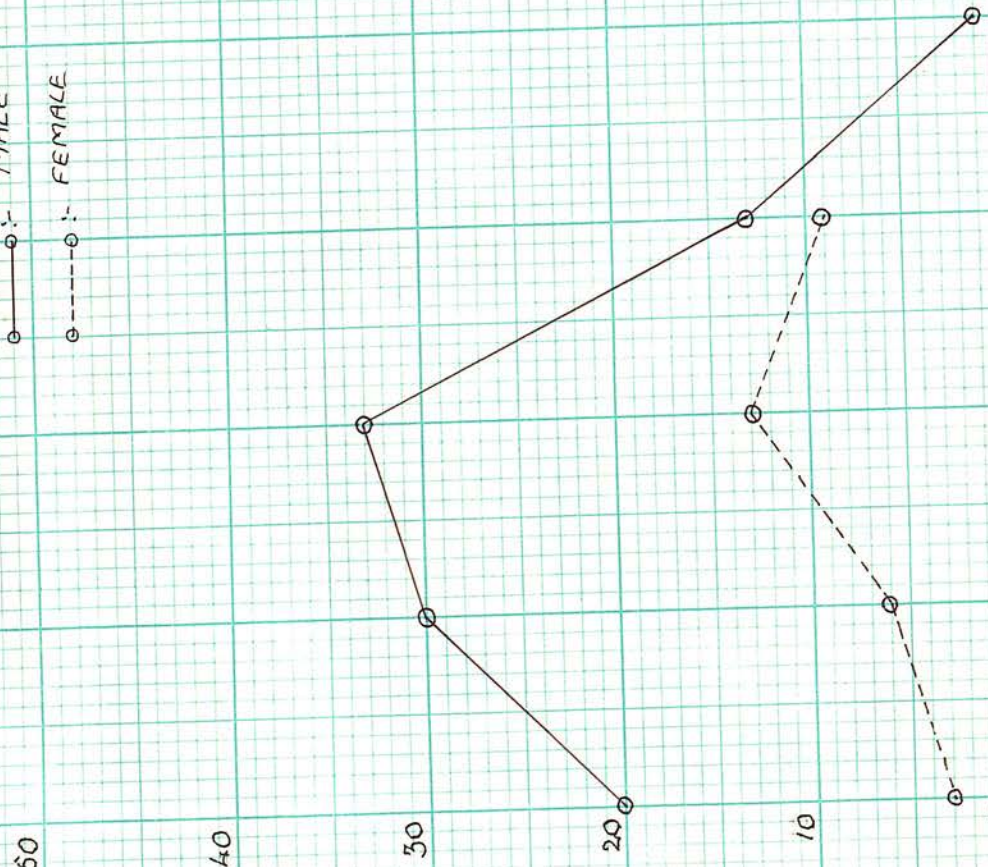
70-79

+ 80

AGE IN YEARS

NO OF CASES

FIG VI.



AGE INCIDENCE.

In this series the average age of all cases was 60 years. The youngest male affected was aged 42 years and the oldest 81 years. The youngest woman with coronary thrombosis was aged 47 and the oldest 79. Cases under 50 years of age were 23 and over 50 years 105, the average age being 58.5 years for males and 64 for females. In most males the attack occurred in the 6th and 7th decades whereas in females the onset was greatest in the 7th. The largest number of cases, however, occurred in the 7th decade between the ages of 60 and 69. 64 per cent of cases were in the 6th and 7th decades.

Table IV *

Age in years	Males		Females	
	No. of cases	Percent- age	No. of cases	Percent- age
40 - 49	20	20	3	9.7
50 - 59	30	30	6	19.5
60 - 69	33	33	13	41
70 - 79	13	13	9	29
80 +	1			
	<hr/>		<hr/>	
	97		31	

The females were five years older than the males at the time of the attack. The females are reported to be on an average from 3 to 6 years older than the males at the time of their onset of the illness.

Coronary thrombosis has always been considered

* Fig:- V, VI.

the disease of the middle aged and elderly, about 90 per cent of the cases occurring in persons between the ages of forty and seventy.

Friedberg's findings correspond with those who revealed a higher incidence between the ages of fifty and sixty. Levine and Brown's (1929) 99 (68.3 per cent) out of 145 cases were in the 6th and 7th decade. The largest number, however, occurred in the 7th decade between the ages of 60 and 69.

Parkinson and Bedford (1928) report a maximum age incidence between 50 and 70 years, though it was fairly common after 40 in their series. Mintz and Katz (1947) found that almost 75 per cent of the women had myocardial infarction between the ages of 50 and 70 years with the greatest number in the 7th decade. About 62.5 per cent of the men in their series had the attack between 50 and 70 years, with the largest number in the sixth decade. Master and his associates (1939) say that approximately two thirds of the attacks in their series occurred between the ages of 45 and 65 and almost one third before 50 years. The peak occurred in the sixth decade. The number of initial attacks rose progressively until the age of 64 after which there was a rapid decrease. Cassidy (1946) reported that about 70 per cent of

all patients in his series were aged between 50 and 70 at onset. 58 per cent of the women and 48 per cent of the men were over 60 at onset. This confirms the general impression that coronary disease tends to become manifest at a later age in women than in men.

When the series are considered separately there is a striking disparity in their respective age incidence. Males invariably show a maximum incidence in the sixth decade. Table V (a) records incidence in the 6th and 7th decades and percentage in the under 60 years and over 60 years group among males.

Table V (a)

Table of age distribution in cases of coronary thrombosis among (a) males

<u>Authors</u>	<u>Incidence in each group</u>			
	6th decade	under 60 years	over 60 years	7th decade
Conner and Holt (1930)	41%	76.5%	23.5%	18.5%
Willius (1936)	41%	62.1%	37.9%	30.6%
Master et al (1939)	35.9%	66.4%	33.6%	26.6%
Rathe (1942)	31%	46%	54%	39%
Katz and Mintz (1947)	32.2%	57.9%	42%	29.3%
Present series	30%	51.5%	49%	33%

The average age of males varied from 54.7 years of Master et al (1939) to the 58.4 of Katz and Mintz (1947).

Females on the other hand generally show a maximum incidence in the 7th decades and it is uncommon for cases to occur in patients under the age of 50 years. Table V (b) gives details of the age incidence in females.

Table V (b).

Table of age distribution in cases of coronary thrombosis among females

<u>Authors</u>	<u>Incidence in each group</u>			
	6th decade	under 60 years	over 60 years	7th decade
Conner and Holt (1930)	46%	65%	35%	22%
Willius (1936)	28.3%	34.8%	65%	41.3%
Master et al (1939)	34.5%	57.5%	42.5%	33.6%
Rathe (1942)	24%	41%	59%	36%
Katz and Mintz (1947)	31.1%	40.5%	59.5%	42.8%
Present series	19.5%	29%	70.9%	41.9%

Conner and Holt (1930) and Master and his associates (1939) do not bear out the maximum incidence in the 7th decade. 65 per cent of 44 cases of Conner and Holt (1930) were under 60 years but the maximum incidence was in the 56-60 year group. Master et al (1939) reporting on 113 female cases found the incidence in the 6th and 7th decades to be practically equal with 34.5% (39 females) and 33.6% (38 females) respectively and the average age 56 years.

As the diagnosis and the knowledge of the

disease has improved so it has been recognised with increasing frequency and the cases are seen in subjects below the age of fifty and forty or even before that age. The occurrence of this condition in young persons, previously reported as an occasional finding, has recently been emphasized by various authors reporting on large series of cases among soldiers between the ages of eighteen and forty. French and Dock (1944) have noted 80 cases of coronary disease in American soldiers aged 20-38 during the recent war. Cassidy (1946) reported that in his series there were 26 males and 3 females at onset under 40. Two males were under 30, the youngest being 26. Newman (1946) has reported on 50 young cases of coronary occlusion from the British armed forces which came under observation during the 2nd world war. 39 of these were fatal (33 dying suddenly) and the diagnosis was established at necropsy. Coronary thrombosis without thrombosis was recorded in 29 of the cases and with thrombosis in 10. The youngest case was 20 and no less than 22 were within the age group of 20-29 years. Moritz and Lamcheck (1946) surveying reports of approximately 1000 sudden deaths from the disease of apparently healthy soldiers between 18 and 40 found that there were approximately 350 sudden deaths from previously

unrecognised heart disease. 300 of these were due to coronary arterio-sclerosis. 8% of 115 soldiers whose deaths were from coronary disease were under 25 years of age and 22% of this group were younger than 30. Poe (1947) reported 9 cases of fatal coronary artery disease in men from the records of an army hospital. These 9 cases comprised 3.2 per cent of all autopsies of a total of 280 performed on men between the ages of 18 and 40 years, and also 7.4 per cent of all autopsies, a total of 121 performed on men between the ages of 18 and 40 years who died of natural causes. Yater et al (1948) studied 866 cases of coronary artery disease in soldiers between the ages of 18 and 39 years inclusive, 450 of whom were examined at necropsy. 64 men of this series were between 18 to 24 years of age, the youngest one being 18. 139 were 25 to 29 years inclusive. 266 were 30 to 34 years of age inclusive and 380 were 35 to 39 years of age inclusive. Rarely proven cases have been observed in childhood and infancy. Today the correct diagnosis is probably made much more frequently for the constant emphasis upon "acute myocardial infarction" in the past decade has made the physician more "coronary thrombosis" conscious.

HYPERTENSION.

Blood pressure was recorded in 88 males and 31 females. It was taken in the wards after the onset of acute coronary thrombosis. An antecedent hypertension was known in 12 patients. All the patients with a systolic pressure greater than 150 mm Hg. or a diastolic pressure of more than 90 mm Hg. were considered to be hypertensive. According to this classification, 60 patients, 40 males and 20 females, a little over one half, had hypertension. The incidence of hypertension was significantly higher among the women (64.5 per cent) than in the men (41 per cent). 21 patients with hypertension died in the hospital. The immediate mortality following coronary occlusion among the hypertensive group was lower (35 per cent) than that of the non-hypertensive group (38 per cent). Hypertension is the most common disease associated with acute coronary thrombosis. The incidence of hypertension in studies of acute coronary occlusion or myocardial infarction as reported in the literature varies from 33 per cent to 73 per cent. In the series of Master et al (1939) 62.4 per cent of their patients had an antecedent hypertension. They thought even this figure was lower than the actual incidence. In the study of Smith, Saul and Bellew (1942) 41 of 100 patients were known to be hypertensive.

Cassidy (1946) states that nearly 70 per cent of his anginal patients without coincident or recent coronary occlusion were hypertensive. He also says that these findings surprised him, for he had not realised that the proportion of hypertensives was so high. In 108 cases of coronary occlusion studied by Fisher and Zukerman (1946) hypertension antedated the occlusion in 65 per cent of the women and 39 per cent of the men. These authors point out that negroes, though more liable than whites to hypertension show a significantly lower incidence of coronary disease.

Studies of the previous histories and post-mortem material, such as heart and kidneys, suggest that the percentage of hypertension in coronary cases appears to be significantly higher than in the general population. Hypertension in women with coronary occlusion is definitely higher than among men with this condition. Levy and Boas (1936) reported that they have seen 1672 women of whom 169, or 10.1 per cent, had coronary artery disease. In the same period they saw 2135 men, of whom 1059, or 49.6 per cent, had coronary artery disease. The frequency is 4.9 times as great in men as in women. Of the 169 cases in women, 125 i.e. 74 per cent were associated with hypertension alone, 25 or 14.8 per cent with both diabetes and

hypertension, and 6, or 3.5 per cent, with diabetes alone. In only 13 cases, or 7.7 per cent, was there neither hypertension nor diabetes, and of these only five were in women under the age of 50. These authors concluded that coronary artery disease is infrequent in women unless there is an associated arterial hypertension or diabetes mellitus.

In the opinion of Fisher and Zukerman (1946) the high blood pressure is not a factor in prognosis. Mintz and Katz (1947) found 35.9 per cent of their cases hypertensive. Hypertension increased with age, especially in women. In their opinion hypertension appears to have no influence on the immediate prognosis of acute myocardial infarction. Rosenbaum and Levine (1941), in their study of 208 cases of myocardial infarction reported that hypertension tended to increase the mortality.

The relationship of hypertension to acute occlusion (coronary thrombosis) is conjectural since neither the ultimate cause of hypertension nor that of arterio-sclerosis is known. It has been suggested that (a) hypertension favours, intensifies or accelerates arterio-sclerosis, (b) similar metabolic, endocrine or nervous factors are responsible for both conditions, (c) simultaneous arterio-sclerosis in the kidney and heart

account for the association of hypertension and coronary thrombosis, and (d) that hypertension predisposes to the latter by favouring the occurrence of intimal haemorrhages in arteriosclerotic plaques.

DIABETES.

In this series 6 patients had sugar in their urine. 2 of them had a transient Glycosuria and the rest diabetes mellitus. The number of male and female diabetics was equal. In one case there was a familial occurrence of diabetes. The patient's father was a diabetic, his sister aged 39 was suffering from diabetes, one brother had sugar in the urine and his mother's ^{daughter} sisters~~s~~ was also a diabetic. Diabetes did not seem so common in this series. Possible explanation of this might be that diabetic patients do not usually come to general ward but go to diabetic clinics and furthermore diabetes being prevalent in certain class and race is found relatively less frequent in the general population. It is also possible that early and efficient modern treatment of the condition might have been responsible for the decrease in number of cases.

Diabetes mellitus is said to be most frequently associated with coronary thrombosis. Root and his associates (1939) made a comparison of atherosclerotic lesions in the coronary arteries of 349 diabetic and 3400 non-diabetic autopsied cases. They observed that coronary occlusion was much more frequent in diabetic than non-diabetic

persons. Between the ages of 40 and 60 the frequency of occlusion was 23 per cent among the diabetics. Between the ages of 60 to 80 occlusions occurred in 43 per cent as compared with 10 per cent among non-diabetics. Antecedent diabetes has been noted in 10 to 23.79 per cent of acute coronary occlusion by various authors. Bean (1937) stated that diabetes was present in 17.4 per cent of 109 patients, but was not characterised by earlier age of onset. Master et al (1939) reported that the incidence of diabetes mellitus in his entire series was 11.2 per cent. Diabetes was found to be more common in the older patients, occurring in 17 per cent of those aged 60 years and over, and in only 4.8 per cent of those under 50. In the series of Mintz and Katz (1947) 16.2 per cent of cases were diabetics. These authors believed that a high level of blood sugar was advantageous.

Glycosuria has been reported frequently in the literature as a sequel of acute myocardial infarction. Levine and Brown (1929) believed it was due to pain and fear during the acute episodes. Edelmann thought that myocardial infarction caused latent diabetes to become manifest. Eppinger (1934) attributed the glycosuria to the absorption of the product of protein destruction which favoured an

outpouring of epinephrine. Raab and Rabinowitz (1936) found abnormal dextrose tolerance curves soon after the acute attack of myocardial infarction which later became normal.

Among elderly diabetics coronary thrombosis is one of the commonest causes of death. Reitman and his associates (1942) found coronary thrombosis in a diabetic youth of 20 years. Like hypertension diabetes mellitus is encountered in a higher percentage of women with coronary occlusion than among men. This has been explained on the premise that arterio-sclerosis advances more slowly in females than in males, and is therefore less likely to be severe enough to result in an occlusion, unless factors like hypertension and diabetes greatly accelerate its progress.

Diabetes was not found as frequent in patients with acute coronary thrombosis as might be judged from the literature.

OCCUPATION.

121 patients whose occupation was known were classed as follows:-

Labourers - (a) skilled 25, (b) unskilled 22.

Housewives - 30.

Retired - 25. This group consisted of all occupations, e.g. labourers, miners, merchants, professionals etc.

Businessmen - 9.

Professionals - 5.

Salesmen - 3.

Executive.

These patients came from an industrial district and belonged mostly to the working classes. There is no occupational distribution and the wards are open to all admissions. No particular inference thus could be drawn from the data in the series as to the etiological relationship between the occupation and occurrence of coronary thrombosis. The disease is found in all walks of life and is not prevalent in any social class or occupation.

Denney (1936) found a high incidence of coronary disease among business and professional classes. Thus of 489 men in his series dying of coronary disease and angina 67 per cent were business men and professionals. He suggests that

the early appearance of sclerosis of the coronary arteries in certain individuals is most commonly attributed to nervous and emotional strain.

Little attention has been given to the fact that the disease is scarcely ever found in men whose occupations require daily physical effort and most frequently in those whose occupations are sedentary. This suggests that physical inactivity predisposes to coronary disease. Rathe (1942) observed that people in rural areas were affected as often as those in urban areas.

Smith, Saul and Bellew (1942) reviewing 100 cases of coronary occlusion noted that 67 per cent of the patients were business men of executive type or those engaged in small businesses which demand excessive individual effort. The frequent occurrence among travelling men was also noted. They suggested that those who work hard and have irregular living habits are more vulnerable to this disease.

RACE.

All the patients in this series were Scots, English and other caucasians. Therefore no comparison with other races could be made. Roberts (1931) found that angina pectoris and coronary thrombosis affect the coloured races less frequently than the white. This fact is of interest as hypertension is much more common in negroes than in the white races; yet the incidence of coronary vessel disease has been shown less in coloured races. Denny (1936) found a high incidence of coronary disease in the Hebrew race. He reports that in Brooklin during 1934 and 1935 of all men dying from coronary diseases 18.8 per cent were Russian Jews. Hedley (1939) in a study of 5116 fatal cases of acute coronary thrombosis in Philadelphia, found the mortality twice as high among the Russian Jews as native born white Americans. This is perhaps due to the well known susceptibility of the Jews to diabetes mellitus. The negro race, which has been considered to have a much lower incidence of coronary thrombosis and about whom Burch (1939) observed that he has rarely encountered angina pectoris in negro males and not at all in negro females in his series, have recently been reported as having only a moderately lower frequency than the white races.



Holoubeck (1945) found 217 deaths due primarily to cardiac arterio-sclerosis in a total of 8,313 autopsies performed. The incidence in the white race was 129 as compared to 88 in the negro race. This group also included 117 cases in which there was evidence of coronary occlusion, myocardial infarction or both. The percentage of occlusion or infarction in the males was 57.3% in whites as compared to 49.9% in negroes.

In the series of Yater et al (1948) there were 850 men of whom 784 were Caucasians, 63 were Negroes, 1 Filipino, 1 Mexican and 1 Chinese. The authors report that since only 10 per cent of the men in the Army were Negroes, the incidence of coronary artery disease in the Negroes was somewhat more than two thirds of that in the white soldiers. French and Dock (1944) in an analysis of the clinical and pathological features of 80 fatal cases of coronary disease in young soldiers revealed that the disease occurred in men of various racial and national origin, showing no predilection for any particular stock.

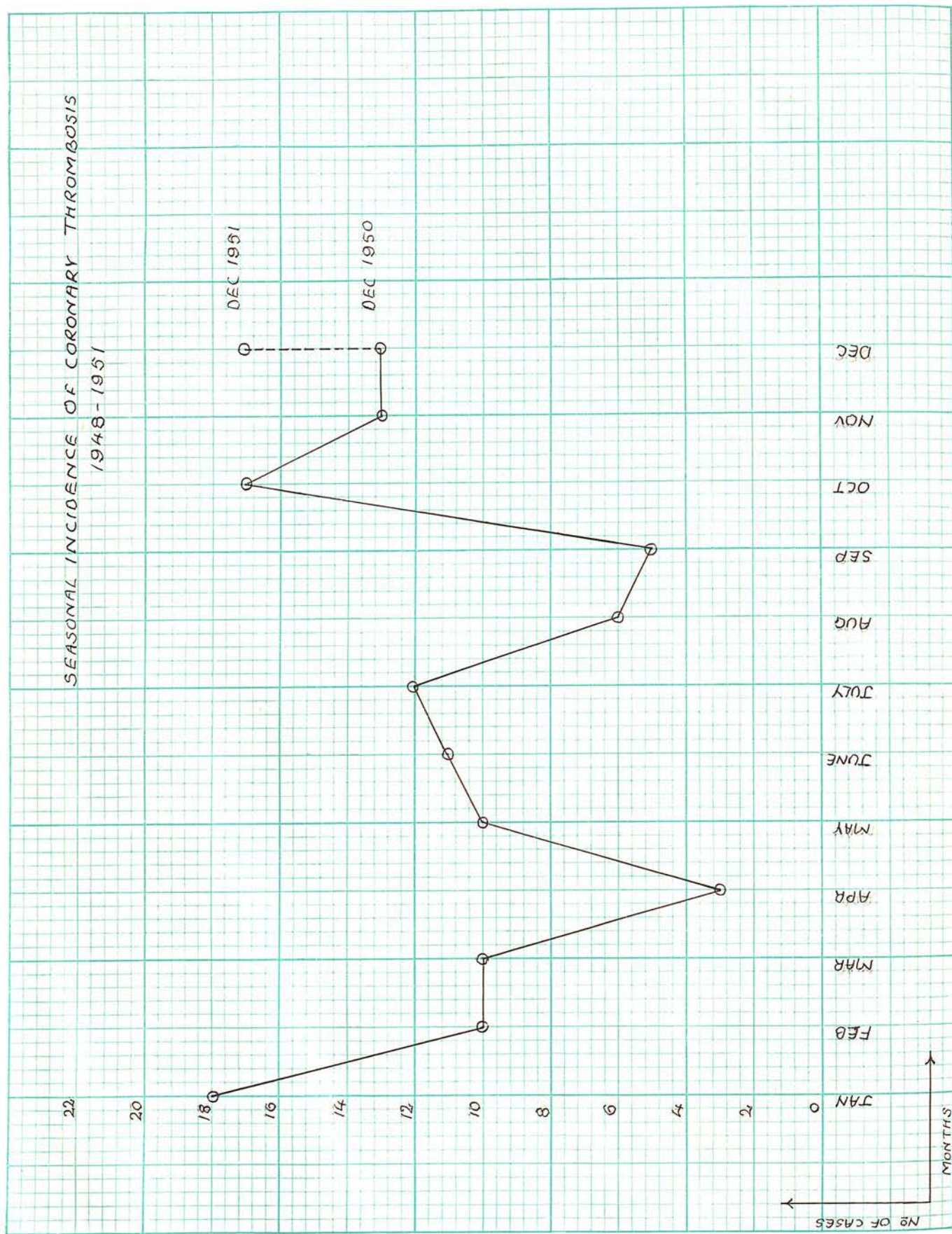


Fig VII

SEASONAL INCIDENCE.

128 cases of coronary thrombosis were admitted to wards 23 and 24 R.I.E. from 1st January 1948 to 30th Nov. 1951. Their dates of admission were analysed. The table given below shows the seasonal incidence of coronary thrombosis in the series. It appears that the majority of incidence of coronary thrombosis took place in the coldest months of the year rather than in the warmest months.

Table VI. *

Seasonal incidence of coronary thrombosis:-

<u>Month of attack</u>	<u>number of cases</u>	<u>percentage of all cases</u>
January	18	14
February	10	7.8
March	10	7.8
April	3	2.3
May	10	7.8
June	11	8.5
July	12	9.3
August	6	4.6
September	5	4
October	17	13.2
November	13	10
December	<u>13</u>	10
	128	

Possible explanation of the variation in the seasonal incidence of the disease might be that upper respiratory tract infections are common during cold and changeable weather and in these infections there is rise in temperature and

* Fig:- VII.

metabolism, which increases the burden on the heart. In stormy weather fluctuations in temperature may produce a strain by vasomotor reflexes, as it is known that angina pectoris is likely to be precipitated by walking in cold or windy weather. Voluntary activity which is increased in the cold may also contribute to the heart burden. All these factors have thus a bearing on the working of the heart and may make the individual more liable to the attack whose heart is already crippled with coronary atherosclerosis.

It may be that the incidence of thrombosis of other vessels show a seasonal variation. Dana says of cerebral vascular accidents that "rather more cases occur in cold weather". Moreover the increased winter and spring incidence of hospital admissions for peripheral arterial disease may not be due to an increased incidence of thrombosis in these vessels during the cold months. There is no seasonal variation in coagulation or other factors which might affect thrombosis.

(1) Wood and Hedley (1935) found a low summer and high winter incidence of coronary thrombosis in 131 cases in Philadelphia between 1932 and 1934 inclusive. Their series included both private and hospital patients.

(2) Bean and Mills (1938) reviewing the series of Master (New York), Mullins (Pittsburg), Bean (Boston), Wood and Hedley (Philadelphia) and Bean and Mills (Cincinnati) concluded that coronary occlusion attacks are definitely more frequent in winter than in summer in the north temperate region.

(3) Hosie (1940) reported that in the sub-tropical climate of Los Angeles, acute attacks of coronary occlusion are definitely more frequent in winter and early spring than in late summer and early autumn.

HEREDITY.

In a series of 128 cases 25 patients volunteered a family history of heart disease, 5 of coronary thrombosis, 3 of angina pectoris and 5 of vascular disease. In all 38 patients in the series had a family history of heart or allied disease, which is a very low percentage, but it is rather difficult to bring out the cause of death of one's near relatives as most of the patients do not remember it and moreover family histories, at no time easy to secure in accurate form, are inevitably defective. The sympathetic consideration often compels the avoidance of detailed enquiries about cardiac illness and deaths in the parents or other near relatives of persons themselves the subject of heart disease. The patients with a family history of coronary thrombosis were of relatively younger age. Their average age was 53 years, furthermore their parents or relations had their coronary thrombosis or died of coronary occlusion at a higher (older) age.

In this group there was a patient whose father and father's two brothers had all died of coronary occlusion. It cannot be mere chance that three members of the same family should die of the same disease. It appears that the original structure that these individuals had at birth must have been

peculiar so that ordinary wear and tear did more harm to certain vessels in them than to other people. It is possible that the anatomic configuration of one of the arteries might have been slightly different so that with repeated contractions of the heart muscles one particular part of the vessel received undue strain, and resulted in its premature degeneration. The peculiarities would be readily inherited just as are queer configurations of the lobes of the ears and other structures. Hereditary susceptibility appears to be a more fundamental etiological factor in the occurrence of coronary sclerosis and occlusion. Hypertension, cerebral haemorrhage or thrombosis and diabetes mellitus appear to occur with more than average frequency in the same families.

Levine and Brown (1929) state that heredity is of considerable importance in the etiology of this disease. Goldsmith and Willius (1937) found that there was a family history of cardio-vascular renal disease in 165 out of 300 cases. Bean (1937) observed that family tendency to cardio-vascular disease was an important factor appearing in 48 per cent of his 138 patients. In the series of Rathe (1942) of 274 patients a definite family history of cardio-vascular disease was

given by 133 patients. Cassidy (1946) states that family history plays a notorious part in the aetiology of cardio-vascular disease and it did so in almost half of his cases.

In clinical practice coronary heart disease is not infrequently encountered in siblings; such observations have led to the hypothesis that coronary heart disease runs in families and Gran and White (1951) considered this question in their study of 100 young candidates for coronary heart disease. These authors found that the incidence of coronary heart disease in the parents of 100 patients and 146 control was 27 per cent and 14 per cent respectively, i.e. twice as many parents of the coronary disease group as compared with the parents of the control group were suffering from heart disease. Another interesting finding in this aspect was that 8.6 per cent of the siblings of patients with coronary heart disease had this disease while only 1 per cent of the siblings of the control group had it.

BODY WEIGHT AND BODY BUILD.

The body weight of 47 patients who died in the wards and were not able to be weighed was not recorded. In the remaining 81, all were not of the same build. Some were thin and under nourished while the others were fat and well nourished. As such the weight of 61 was recorded. Of these 15 males and 4 females were overweight, according to the standard ratios of height and weight and thus 28 per cent of patients in the survival group were overweight. This figure of 28 per cent for overweight patients in a Series of 128 cases with myocardial infarction is therefore not statistically significant.

Obesity has often been mentioned as a predisposing factor. In the series of Baker and Willius (1938) in 77 cases the ratios of height and weight were recorded. In 87 per cent of these cases varying and definite degrees of obesity were found. French and Dock (1944) observed that 73 out of 80 young men who succumbed to coronary disease were overweight. Yater et al (1948) reported only a slight tendency for men with coronary thrombosis to be overweight. There seems to be no doubt that obesity is a very undesirable status in heart disease and of itself is capable

of swinging the balance from cardiac competence to incompetence.

Patients with acute coronary thrombosis have been described as conforming to a definite constitutional type, characterised by a short stocky stature, some obesity, a thick short neck and a barrel-shaped chest. There is very little scientific information regarding an association between specific types of body build and the occurrence of coronary thrombosis.

Gertler and associates (1951) in their study of young candidates for coronary heart disease prior to the age of 40, have reported a profile pattern. The coronary profile pattern according to these authors consists of:- 1. Hereditary history, (a) presence of coronary heart disease in any sibling at an early age (under 45), (b) history of metabolic disease such as diabetes and xanthomatosis, or degenerative diseases such as gout and arthritis, (c) history of hereditary hyperuricemia and hypercholesterolemia.

2. Psychological history:- (a) strong goal-directed drives, usually with accomplishment, (b) "aggressive" pattern, not an outstanding feature, (c) high athletic rating on contact sports, (d) "less masculinity" in absolute values as revealed by the Terman Miles test.

3. Anthropometry and Somato type:- (a) dominant mesomorphy; secondary dominance endomorphy, (b) dominant ectomorphs the least susceptible, (c) decreased linear measurements:- stature, total face length, hand length, sternum ensiform and span, (d) increased horizontal measurements:- shoulder breadth, upper chest depth, bipupillary measurements, nose breadth, bigonial measurements, head breadth, hand breadth, (e) no evidence of abnormal weight increase in patients with coronary heart disease, (f) tendency to lowering of ponderal index (height over the cube root of weight) in coronary heart disease group.

4. Biochemistry:- (a) serum cholesterol level elevated, (b) serum uric acid level elevated, (c) serum lipoid, phosphorus value slightly elevated, (d) serum cholesterol/serum lipoid phosphorus ratio increased, (e) C.U.P. index $\left(\frac{\text{cholesterol} \times \text{uric acid}}{\text{lipoid phosphorus}} \right)$ increased, (f) reducing intensity of saliva to low emf values, (g) urinary 17-Ketosteroid excretion, low normal, (h) basal metabolism rate, low normal.

5. Clinical history:- usually no signs prior to the coronary episode.

TOBACCO.

Out of 128 patients 34 were heavy smokers, 17 light smokers and the rest did not smoke at all. There is no evidence that excessive smoking develops coronary disease or acute occlusion. Tobacco has long been thought to be a factor in the causation of arterial spasm, and Stewart et al (1945), while studying the effect of smoking standard cigarettes, have demonstrated that it usually lowers the peripheral skin temperature, diminishes the peripheral blood flow and raises the systolic and still more the diastolic blood pressure. Roth et al (1944) also reported that even smoking of two standard cigarettes or the intravenous injection of 2 mg. of nicotine would cause the above phenomenon. It is said that these changes may be evident not only during the smoking of the cigarettes but also sometimes for as long as 30 minutes.

Smoking therefore has been implicated because (1) it has been proved harmful in thrombo-angitis obliterans, another arterio-sclerotic disease, (2) of the vaso-constrictor effects of smoking on the peripheral circulation, and (3) in some patients it seems to precipitate attacks of angina pectoris. There is insufficient evidence of casual relationship between smoking and coronary thrombosis.

ALCOHOL.

Out of 128 patients 4 patients were heavy drinkers, 20 moderate drinkers, and the rest did not drink alcohol at all. Alcohol, like tobacco, has no significant casual relationship in the development of coronary thrombosis. Alcohol has long been viewed with favour as a therapeutic agent in patients with coronary sclerosis and angina pectoris, although its pharmacological effect on the coronary circulation is uncertain. It is said to produce vasodilatation. It is a common opinion that the use of alcohol may help to prevent angina pectoris and that the apparent increase of angina pectoris in United States of America during the past decades may be in part the result of prohibition.

Leary (1931) has found relatively little aortic sclerosis in alcoholic individuals. Cabot (1904) found that only 6% of 283 patients with chronic and excessive alcoholism under 50 years of age showed any clinical evidence of arterio sclerosis while 13 per cent of 45 patients with arterio sclerosis gave any history of alcoholism. Of 656 autopsy cases of arterio sclerosis in his series only 95 were under the age of 50. Out of these 95 in which arterio sclerosis was found at postmortem only 20% and if cases complicated by chronic nephritis were excluded only 17% appear to

have consumed alcohol in any notable excess.
There is thus no causal relationship between
alcohol drinking and coronary thrombosis.

VII. PRECIPITATING FACTORS.

MODE OF ONSET AND TIME OF ONSET.

In this series 102 patients were suddenly taken ill, while in 26 the episode developed gradually. In 82 patients the incidence took place during the daytime and in 46 during the night. A day is considered to be from 6 a.m. to 6 p.m. 59 patients were resting in bed, armchair or in the house, while 23 were sleeping. Mild activity which included routine housework, walking in or out of the house attributed to 16, while moderate activity such as a brisk walk, running after a tramcar and other moderate exercise was associated with 24 attacks. Vigorous activity of the type of strenuous exercise or work like boring a hole in a wall, or lifting up heavy weights, etc., caused the attack in 6 patients.

Table VII.

Physical activity at onset of attack.

<u>Activity</u>	<u>No. of cases</u>	<u>Percentage incidence</u>
Sleeping	23	17.95
Resting in bed or armchair etc.	59	46
Mild activity	16	12.5
Moderate activity	24	18.5
Vigorous activity	6	4.6

The table shows that in the majority of cases (64 per cent) the acute episode of coronary occlusion occurred during complete inactivity.

The incidence occurred during mild activity in 12.5 per cent, and during moderate or vigorous activity in 23 per cent of the cases. A history of usual routine of day to day life was obtained generally. The majority of incidence occurring at the non-activity period seem to be due to the circulation being at its slowest rate.

The possible factors in the diminution of coronary flow are:- (a) lowering of blood pressure, (b) lessening of cardiac output and (c) coronary constriction by vagus stimulation. It might be suggested that rest is more favourable for the formation of thrombi than activity.

Grollman (1930) found that the blood pressure, both systolic and diastolic, and the cardiac output in sleeping subjects fall progressively until about 4 a.m. Whether this is due to the prolonged rest or to the low phase of diurnal rhythm is not known.

Phipps (1936) in a study of 437 cases of coronary occlusion found physical exercise responsible in 13 per cent, moderate or usual exertion in 18 per cent while 51 per cent of the cases were at rest and 8 per cent asleep. Master, Dack and Jaffe (1937) revealed that among 530 attacks of coronary thrombosis 43.1 per cent occurred while the patient was asleep or resting,

13.68 during mild routine activity, 18 per cent while the patient was walking and 5 per cent during moderate activity. In only 2 per cent was there unusual physical exertion. The authors concluded that the percentage of attacks which occurred during sleep, rest or mild, moderate or intense activity, coincided with the proportion of the day spent in these states and that physical exertion, which is an important precipitating factor of angina pectoris is not concerned in the initiation of acute coronary thrombosis.

Fitzhugh and Hamilton (1933) noted the occlusion occur 24 times during or following some unusual and violent exertion, 31 times after an unusual one and 44 times after an unusually prolonged usual exertion, including loss of sleep and rest resulting in unusual fatigue in their 100 cases. Boas (1939) reported 14 out of 25 cases of cardiac infarction following an unusual effort which had come under his personal observation. Cooksey (1939) found that 17 out of his 100 private cases of acute coronary occlusion had developed within 24 hours of definitely unusual and frequently rather extreme physical exertion. Smith and associates (1942) found physical exertion associated with 32 of 51 cases of coronary occlusion.

In the Series of French and Dock (1944). In 15 cases sudden death or the onset of severe pain in a fatal seizure occurred during "vigorous exercise" or violent muscular effort. 26 patients or 36 per cent had the fatal attack within one to several hours after "vigorous exercise", 5 of these dropped dead and 4 died in sleep. Yater and associates (1948) reported that the onset of the "coronary attack occurred relatively more often during strenuous activity than during mild and moderate activity and sleep. The proportion of attacks among young soldiers occurring during strenuous activity was more than twice as great as the proportion of time spent in such activity, the proportion during mild and moderate activity was about equal to the proportion of time thus spent and the proportion of men stricken while asleep was about one third that of the proportion of time normally spent in sleep. The exertion reported as unusual and severe by these various authors included cranking a car in cold weather, lifting a heavy trunk, pushing a stalled car, rowing, long hikes uphill etc. Fitzhugh and Hamilton emphasized that the effort had often been performed when the subject was suffering from undue fatigue, inadequate sleep and excessive emotional strain. While observations of Phipps and Master et al might

indicate that physical exertion is not an essential or even important precipitating cause in most cases of acute coronary thrombosis, it does not exclude the possibility that it induces at least some instances of coronary thrombosis.

It is also difficult to see why myocardial infarction should sometimes occur at the height of severe exercise, when the conditions present in the coronary circulation must be those least likely to produce organic occlusion. Two possible explanations are put forward (1) It may be that the increased blood flow tears off an atheromatous plaque which obstructs an artery distally.

(2) It might be suggested that if at the height of a severe anginal attack a generalised vasoconstriction through the coronary circulation does indeed occur, it might persist for an abnormally long time and permanent changes in a portion of the heart muscle supervene.

OPERATION.

In this series three of the patients had their occlusion after an operation. In the first case occlusion occurred two months after a gall bladder operation; in the second in a convalescent home while dressing a colostomy and in the third one, one week after the removal of a suprapubic cystostomy tube. Attacks of acute myocardial infarction soon after operations have been observed with sufficient frequency to suggest that there is a significant causal relationship between the operation and the acute infarction.

Rendall and Orr (1930) reported two cases of coronary occlusion following operation occurring in the surgical clinic of the University of Kansas. Menard and Hursethal (1931) observed coronary occlusion in three patients after the operation. Master and his associates (1938) noted that 5.6 per cent of 625 attacks of coronary occlusion followed an operation. The persons affected as a rule are beyond the age of 50. The rise in blood platelets and fibrinogen after the operation, combined with surgical shock, slowing of circulation by bed rest or lessened activity, attended by a diminution in blood volume and a drop in blood pressure, may be attributed the possible factors associated with operation leading to coronary occlusion.

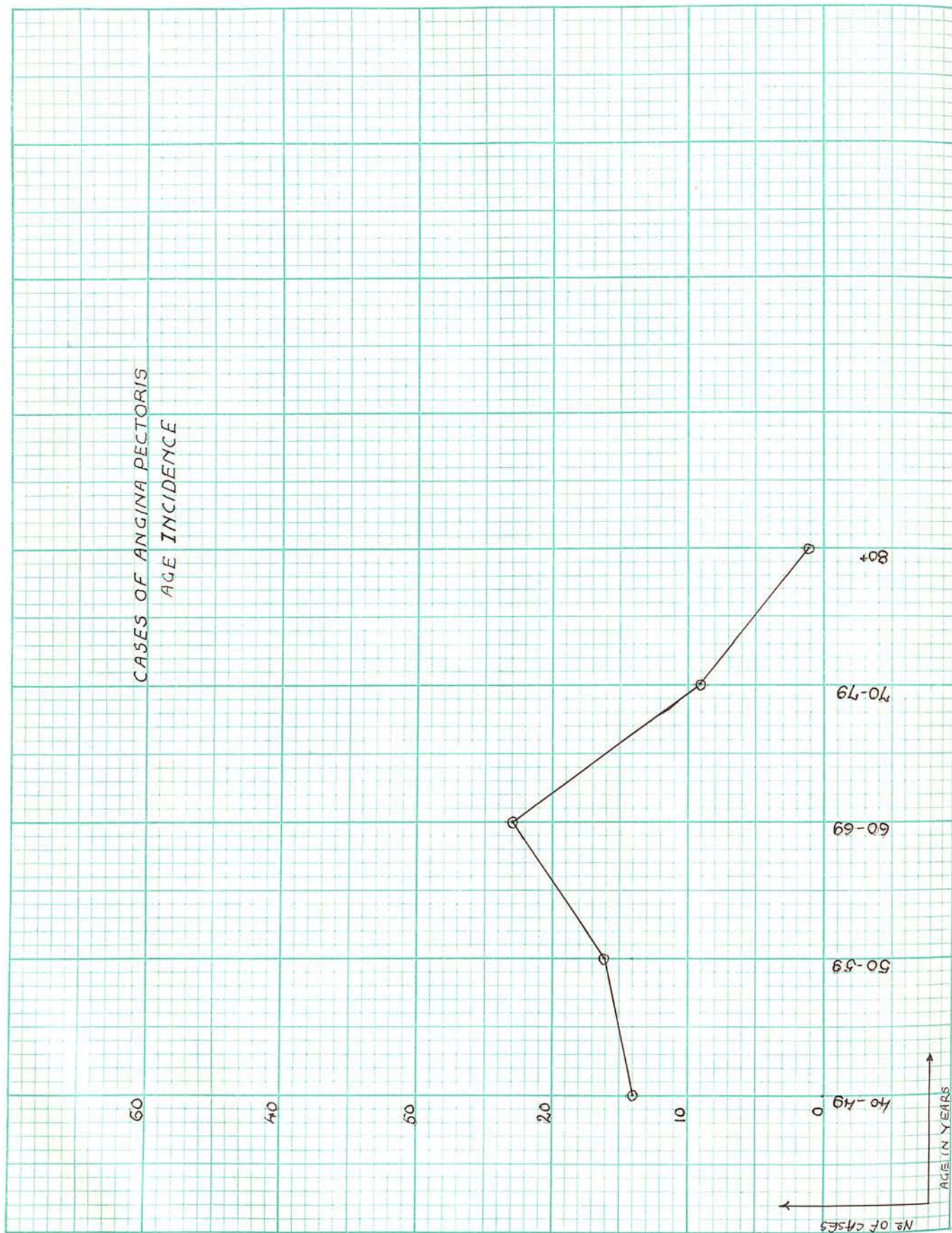


Fig. VIII

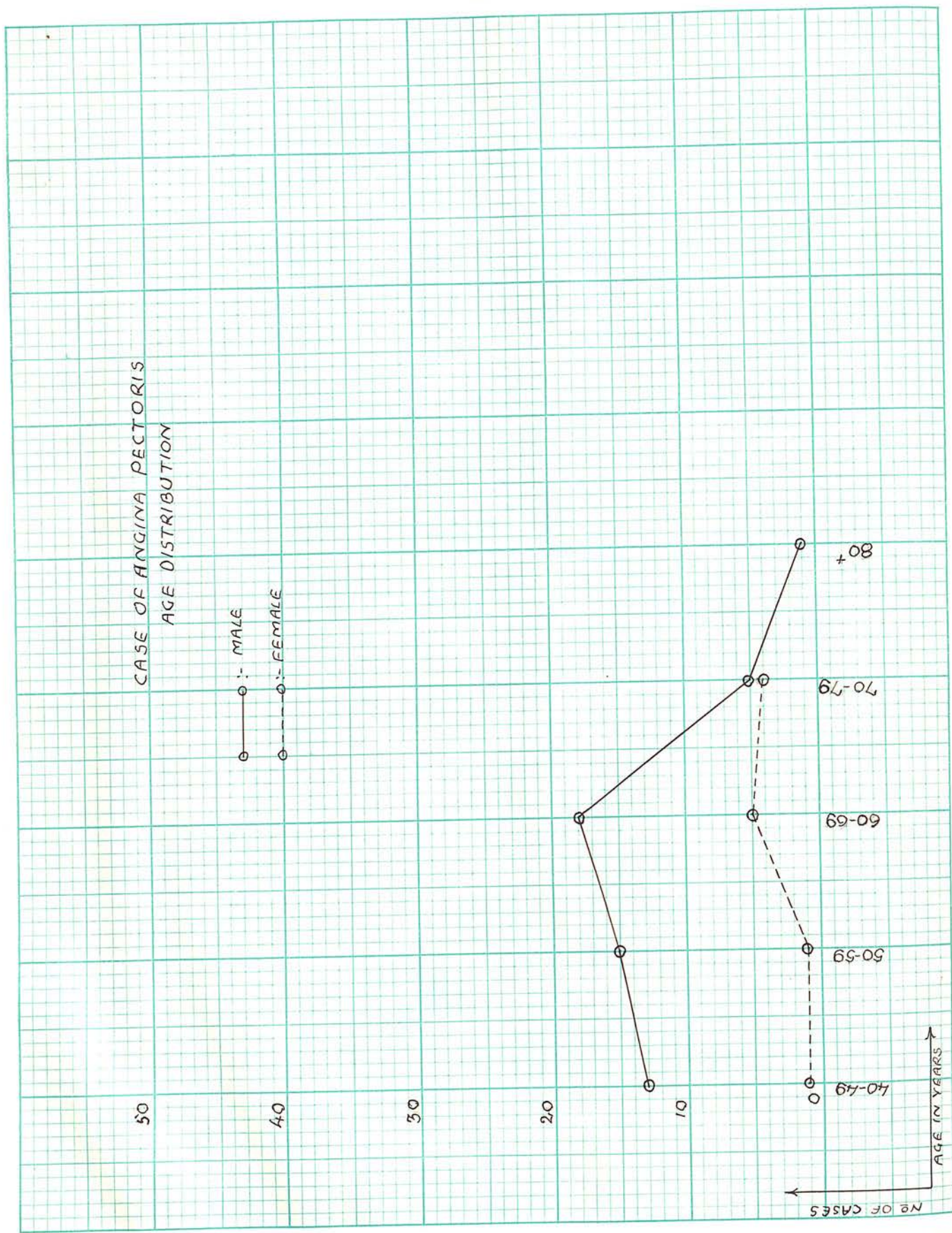


FIG VIIIa

ANGINA PECTORIS.

In this series 63 patients, 52 male (53.6 per cent) and 11 females (35.5 per cent) had a previous history of angina pectoris, thus giving a sex ratio of 4.5 males to 1 female. The average ages of males and females were 58.25 and 65.5 years respectively i.e. about the same as those of the whole series, 58.5 and 64 years. 19 males and 2 females died and averaged 61.7 and 70 years respectively. The overall mortality in this group was 33.3 per cent, while the immediate mortality in the rest of the series was 40 per cent.

Table VIII. *

Showing the age incidence.

Ages in years	<u>Males</u>			<u>Females</u>		
	Total No. of cases	No. of angina cases	Percent- age	Total No. of cases	No. of angina cases	percent- age
40-49	20	13	25	3	1	9
50-59	30	15	29	6	1	9
60-69	33	18	34.6	13	5	45.5
70-79	13	5	9.6	9	4	36.36
80+	1	1				
	97	52		31	11	

The women showed a higher incidence in the higher age group. 9 of 11 women had an antecedent hypertension. All the patients with angina pectoris were beyond the age of 40 and about 80 per cent were over 50.

* Fig:- VIII, VIIIA.

The immediate mortality for patients with angina pectoris was less than those who had not this symptom. Chambers (1946) found the immediate mortality the same in both anginal and non-anginal cases. Fisher and Zukerman (1946) reported a less favourable prognosis for patients giving a history of angina pectoris where as Rosenbarum and Levine (1941) observed, angina pectoris in the past improved the outlook of the individual with an acute myocardial infarction. Glendy and associates (1937), while discussing coronary artery disease in patients under 40 years of age, pointed out that a past history of angina pectoris improves the prognosis of occlusion.

Angina pectoris occurs predominantly in males, the ratios varying from 3 males to 1 female to 6 males to 1 female as reported in the literature.

In the series of Parker et al (1946) there were 2,794 men and 646 women, giving a ratio of 4.3:1. Boas (1936) found coronary artery disease 4.9 times as frequent in men as in women. Eppinger and Levine (1934) studied 141 cases of angina pectoris and there were 30 women and 111 men in their series.

90-95 per cent of patients with angina pectoris are generally beyond the age of 40 and more than 70 per cent are beyond the age of 50. Glendy et al (1937) have however observed angina

pectoris in persons below the age of thirty.

Eppinger and Levine (1934) found the average age of onset of angina pectoris to be 56 years in males and 58.1 years in females. The range is between 35 and 70 years.

Pre-existing angina pectoris has been reported in from the 22.4 per cent of Willius (1936) to the 72.9 per cent of Katz and Mintz's (1947) series. The incidence varies depending on the completeness of the case histories. Table IX. gives the incidence of angina pectoris preceding acute coronary thrombosis as reported by various authors.

Table IX.

Incidence of pre-existing angina.

<u>Authors</u>	<u>Incidence of antecedent angina pectoris</u>
1. Parkinson & Bedford (1928)	62 per cent
2. Conner and Holt (1930)	26.1 " "
3. Willius (1936)	22.4 " "
4. Bean (1937)	33.6 " "
5. Rosenbaum and Levine (1941)	72 " "
6. Bland and White (1941)	62.5 " "
7. Fisher and Zukerman (1946)	41.66 " "
8. Katz and Mintz (1947)	72.9 " "
9. Present series	49.2 " "

Roberts (1931) found angina pectoris a rarity in negroes, but Weiss (1939) reported that this rarity is becoming more common in recent years as the negro has become more exposed to the economic and emotional strains of industrial life.

Heredity is an important predisposing factor of the disease. A careful familial history when this is known to the patient usually discloses that the patient's parents, siblings etc. also suffered from angina pectoris or other incidence of coronary arterio-sclerosis. Family history of angina pectoris was obtained from three patients in this series. In one case, mother, sister and a brother all died of angina pectoris. Angina pectoris may appear in persons of any occupation, there is a strong clinical impression that it affects particularly those whose work subjects them to continuous mental tension.

Paul D. White says of angina pectoris - "It is encountered more in communities where the strain of life is great and a hurried existence a habit."

Sir William Osler (1897) says of angina pectoris "It is not a disease of the working classes. The life of stress and strain, particularly worry, seems to predispose to it and this is perhaps why it is common in our profession."

Hypertension is often found in patients with angina pectoris and is especially frequent in women. As a rule angina pectoris occurs in females when there is an associated hypertension or diabetes. In the series of Eppinger and Levine (1934) the average reading in men was 149.2 systolic

and 89.2 diastolic and the corresponding figures in women were 190 systolic and 102 diastolic. There were no women with a systolic reading under 140 mm. Hg. All but 7.7 per cent of 169 women in the series of Levy and Boas (1936) had either hypertension or diabetes mellitus. Summers (1948) found 84 per cent of women suffering from hypertension in his 87 cases of angina of effort occurring in women. Hypertension would appear to be a more important factor in the aetiology of angina pectoris in women. Moreover the importance of hypertension in the diagnosis of this condition in women should be stressed.

From an analysis of the anatomic observations in a group of cases of angina pectoris associated with hypertension and a group without hypertension, Davis and Klainer (1940) found that an extreme degree of coronary disease involving two or more major arteries was present in 95 per cent of the patients without hypertension and only in 39 per cent of the patients with hypertension. They concluded that if hypertension is present angina pectoris occurs frequently even in the absence of severe disease. But in subjects with normal pressure angina pectoris is almost always dependant on the presence of severe coronary disease.

In this series 14 patients had a previous history of coronary thrombosis. Of these 9 subsequently developed angina pectoris. In other words 9 patients developed angina pectoris after their first attack of coronary thrombosis. Much more often attacks of angina pectoris, which are absent before the attack, develop after recovery from the acute infarct. Palmer (1937) and Master et al (1942) observed angina pectoris in about 60 per cent of the patients who have recovered from an acute cardiac infarct. Bland and White (1941) found that 39 per cent of their 162 patients who had recovered from the acute attack were limited in their activity by angina.

Smith, Sauls and Ballew (1942) studying 100 patients with coronary occlusion noted that 33 had angina pectoris before and after their attack. 23 patients complained of angina pectoris after myocardial infarction. 13 patients noted that their anginal attacks disappeared after the occlusion.

The high incidence of preceding angina pectoris indicates that from 30 to 40 per cent of all patients with this affliction will eventually succumb to an attack of coronary thrombosis.

Levine (1950) writes that coronary thrombosis is related to angina pectoris in much the same way

as the occlusion of a vessel of the leg with gangrene is related to intermittent claudication. Anginal state may be regarded as a transitory one leaving the heart in practically the same condition after an attack as before. When partial or complete occlusion occurs, the muscles supplied by that vessel suffer to a lesser or greater degree. Sometimes during the life of those suffering from angina, a thrombosis of a coronary artery is apt to occur. This is the commonest cause of death in angina although not a necessary one.

The pathological basis of angina pectoris has been suggested to be anoxaemia of the myocardium no matter how it is brought about. This explains the overwhelming frequency of angina pectoris in the coronary arteries disease. It also accounts for its occurrence in some cases of anaemia with comparatively normal arteries, for anoxaemia of the heart might well result from the lack of haemoglobin. It also accounts for the disappearance of angina pectoris in some cases of thyrotoxicosis when the demands on the heart are decreased by diminishing the body metabolism.

Recently, however, considerable doubts have been thrown on this belief by the experiments of Green and Gregg (1940) and Green and Wegria (1941). The workers demonstrated that in anaesthetised

animals the production of an area of myocardial ischaemia is followed by a great increase in total coronary blood flow. The work of Manning, McEachern and Hall (1939) and LeRoy, Fenn and Gilbert (1942) suggests that these results are not applicable to conscious animals and it may well be that anginal pain in man is followed by a generalised coronary vasoconstriction brought about by parasympathetic stimulation.

It is conceivable that fatal angina might occur under unusual circumstances with structurally normal coronary arteries. Men working with nitroglycerine are known to drop dead instantaneously on slight effort after being away from their work for a few days. These observations have been made by Cecil K. Drinker, and lend support to the theory that the mechanism of coronary spasm is a cause of angina.

The relationship of angina pectoris to coronary thrombosis. In this series there were 63 cases in which a previous history of angina pectoris was found. 9 of these patients had angina pectoris after a previous attack of coronary thrombosis, so there were 54 cases really in which coronary thrombosis had developed after an antecedent angina pectoris. Similarly in the remaining 65 cases in the series there had been no antecedent angina

pectoris and the episode had occurred like a bolt from the blue. In order to establish relationship between coronary thrombosis and angina pectoris both of these groups i.e. cases with an antecedent angina pectoris and cases without an antecedent angina pectoris were analysed and the findings tabulated as under:-

Table X. Showing the comparison of coronary thrombosis and angina pectoris.

	Angina pectoris	Coronary thrombosis
No. of cases	54	65
Age	The average age for males 58.37 yrs. and females 65.5 yrs. Incidence was higher in 6th and 7th decades in men and 7th decade in women.	The average age for males 59.5 yrs. and females 63.45 yrs. Incidence was higher in 6th and 7th decades in men and 7th decade in women.
Sex	43 males (79.6%) and 11 females (20.30%).	45 males (69.27%) and 20 females (30.7%).
Family History	3 patients had a family history of angina pectoris.	5 patients had a family history of coronary thrombosis.
Diabetes	1	3
Hypertension	26	28
Died	18	26
Recovered	36	39
Overweight	5	13
State of coronary arteries	Atheromatous changes were found in 3 of the 14 autopsied cases.	Atheromatous changes were found in 8 of the 14 autopsied cases.
Type of infarction	18 anterior type, 11 posterior type and 4 antero-posterior type.	28 anterior type, 17 posterior type and 4 antero-posterior type.
Complications after the infarction	Embolization, 2. Left ventricular failure 1. Congestive heart failure 2.	Embolization 3. Left ventricular failure 8. Congestive heart failure 8.

The subject of coronary thrombosis has been investigated by various authors and the outcome of their valuable observations throw ample light on the origin of anginal pain. It has been observed that a severe and fatal infarction may not cause pain in rare cases but there is generally a feeling of tightness or constriction varying from a sense of pressure to an intolerable pain in the Substernal region. There is so much in common between this sensation or constriction and the pain in the anginal attack which is evoked by effort and subsides by rest that they are generally regarded to be the different phases of the same process. The relationship is however not so clear because all the cases who develop coronary thrombosis do not have pre-existing anginal pain nor those who suffer from anginal attacks ultimately develop coronary thrombosis. Majority of patients with coronary thrombosis have had attacks of pain resembling more or less typically to angina pectoris. In the Series of Parkinson and Bedford (1928) only 38% had had no previous angina. Levine (1929) reported that majority of patients in his Series had definite angina pectoris ante dating the attack. The close relationship between coronary occlusion and angina pectoris is further shown by the high incidence of occlusion in patients with angina

pectoris.

The object of the analysis in this Series was to find how far the two Syndromes i.e. coronary thrombosis on one hand and angina pectoris on the other were related.

The first method of comparison adopted was of determining the age and sex incidence in the two conditions.

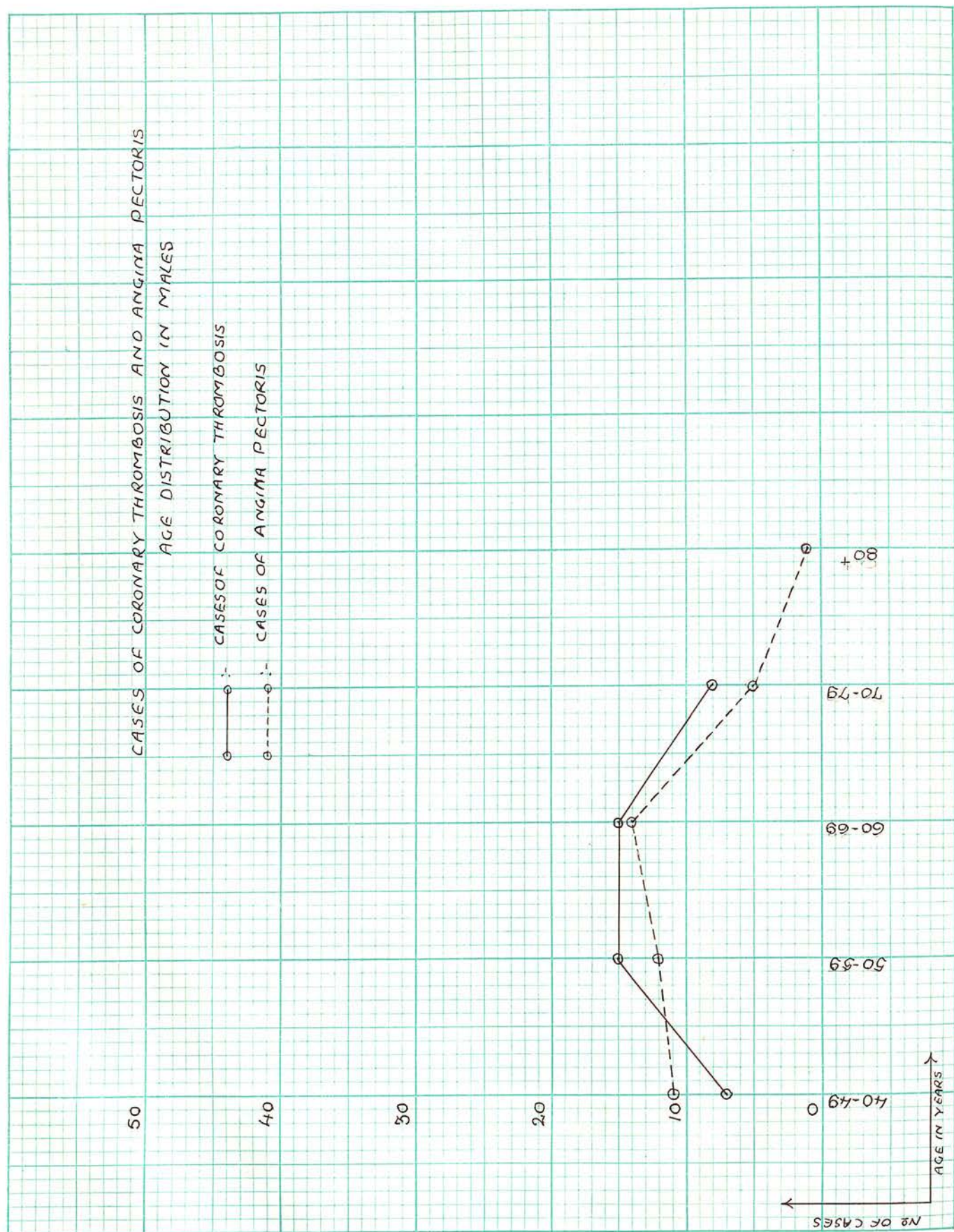


Fig IX

CASES OF CORONARY THROMBOSIS AND ANGINA PECTORIS

AGE INCIDENCE IN FEMALES

--- CASES OF CORONARY THROMBOSIS

--- CASES OF ANGINA PECTORIS

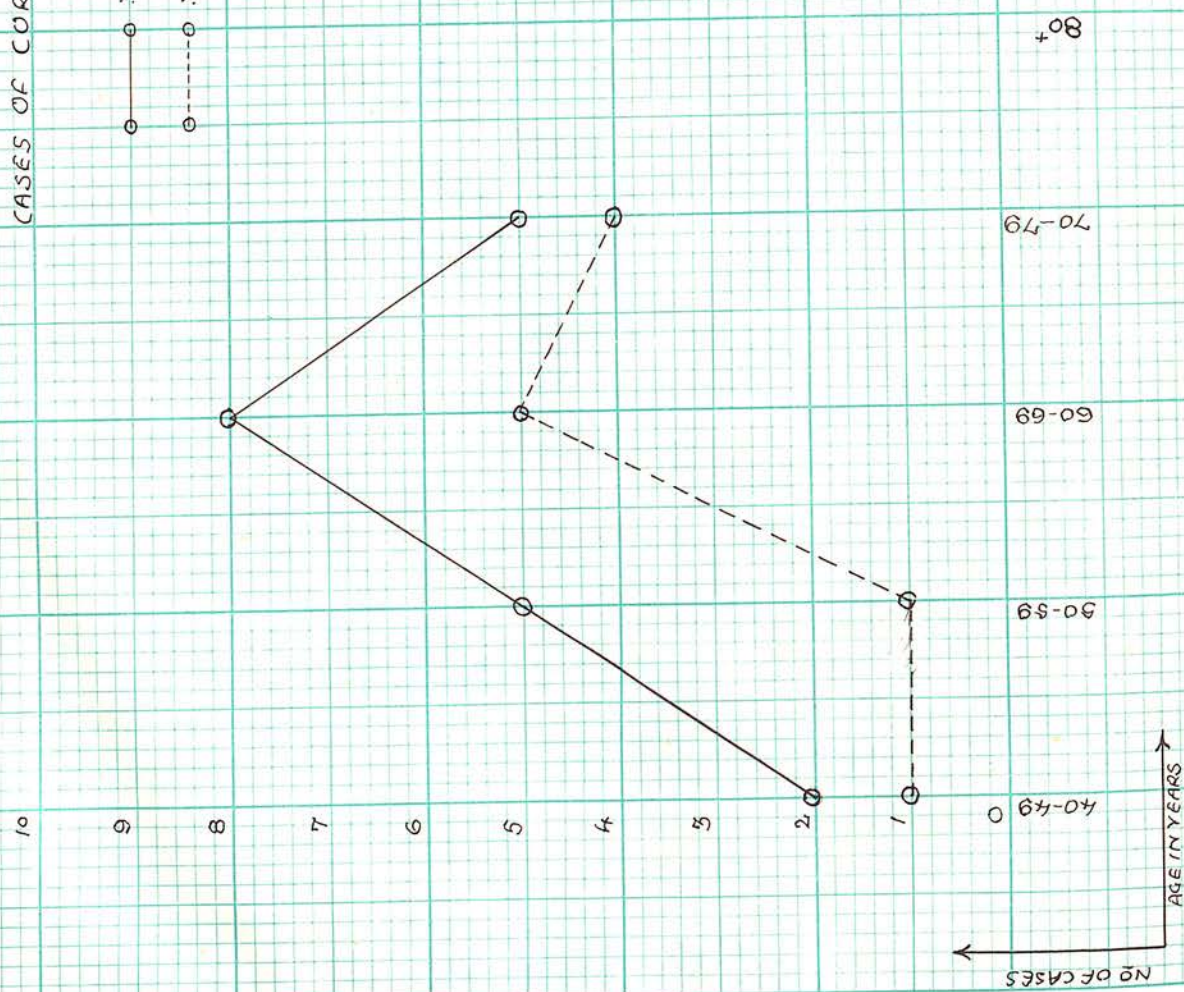


FIG X

Table. XI. *

Showing the age and Sex incidence in Coronary Thrombosis and Angina Pectoris.

Age in years	Coronary thrombosis No. of Male	Angina pectoris No. of Male	Coronary thrombosis No. of Female	Angina pectoris No. of Female
40-49	7	11	2	1
50-59	15	12	5	1
60-69	15	14	8	5
70-79	8	5	5	4
80+		1		
	45	43	20	11

82.

* Fig:- IX, X.

The perusal of the table and the accompanying graph will show that there is a close resemblance between the two conditions. The episode of coronary thrombosis and angina pectoris in men was greater in 6th and 7th decades where as in women the occurrence of both the conditions was greatest in 7th decade. If the sex incidence of both the conditions in the two groups is compared it will appear that more men than women were victims of these ailments.

Heredity factor of both angina pectoris and coronary thrombosis is well recognized and there are striking examples, best known of which is the Arnold family. In the present study also three patients gave a family history of angina pectoris and five of coronary thrombosis. In one instance, mother, sister and a brother all had angina pectoris while in the other patient's father and father's two brothers had died of coronary thrombosis. The possible explanation of this familial incidence may be that some families are more liable to arterial degeneration and this liability is apt to be particular as well as general, since it includes a predilection for certain vessels, e.g. families whose members die of cerebral haemorrhage. It may be suggested that aberrations of coronary arteries as pointed out by

Hadfield may throw an undue measure of responsibility on the one trunk, may run in families.

Hypertension is another factor that is common in both the conditions and is an aetiological factor. In the series 26 patients in the anginal group and 28 in coronary thrombosis group had hypertension. Both angina pectoris and coronary thrombosis occur in patients with high arterial tension, senile degeneration of heart, coronary disease and are rare in infective and toxic diseases of the heart. The anginal pain is therefore produced by those conditions or types of disease in which there is a failure in supply of oxygenated blood to the heart muscle.

The pathology of two conditions is also the same i.e. the Ischemia of the heart muscle. In the case of angina pectoris the ^{temporary} ischemia is reversible while in the case of myocardial infarction it is otherwise and the damage done to the tissues is of permanent nature. The cause of ischemia excluding the precipitating factors in both the conditions lies in the coronary arteries. In this series 3 of the 14 autopsied cases in the anginal group showed atheromatous changes in the coronary arteries. These changes were more or less uniform resulting in the narrowing of the lumen and calibre of these arteries. Similarly there were 8 of the 14

autopsied cases in coronary thrombosis group which also showed atheroma in the coronary arteries. The atheroma here was more of a patchy type and less uniform. The narrowing of the lumen was also less marked than that of the anginal group. This difference in the distribution of atherosclerosis in the coronary arteries in the two groups may be the cause of gradual onset, preceded by anginal pain in one and sudden occlusion like the bolt from the blue in the other.

The changes in coronary circulation demonstrated by injection methods have revealed that as the age advances an encroachment on the lumen of coronary vessels, resulting in narrowing of the calibre on one hand and increased vascularity of the subepipericardial fat on the other takes place. The narrowing thus produced causes serious reduction of the coronary circulation which subjects the cardiac muscle to an urgent need for anastomosis (collateral circulation) by which the diastolic flush of blood may be maintained in spite of obstruction in the main trunks. The progress of these changes correspond so closely with increasing incidence of anginal attacks that it seems justifiable to regard them closely related. The attacks of angina pectoris may be produced by factors other than arterial, evidence shows that

anoxaemia of heart muscle resulting from deficient circulation is the basic factor in the majority of cases and this deficiency of the circulation in most of the cases results from coronary sclerosis. The various theories advanced to explain the causation of anginal pain cannot be discussed here but it is believed that the study of coronary thrombosis has nailed angina for ever to the tree of decaying coronary arteries.

The production of thrombosis depends upon the condition of the vessel wall, and possible slowing of circulation with change in the character of blood. Anything that might therefore lessen the coronary flow or change the condition of the blood so as to make it more coagulable might precipitate occlusion. Increase in need for oxygen with decrease in coronary flow does not favour occlusion, nor does increased oxygen-need with increased coronary flow, both of these conditions may produce angina. Embolism is a rare cause of coronary occlusion. The lesions of intima are almost always present in patients who experience this episode, study of contributory factors as such becomes essentially a study of those factors which diminish coronary circulation.

The attack of coronary thrombosis rarely occurs during unusual effort or excitement, conditions

which usually produce angina pectoris. In this series in 18% of patients pain started while they were asleep or in bed often in the early hours of the morning and in 46% during complete inactivity. The pain began during mild activity in 12.5% of patients and in 18.5% and 4.6% during moderate and vigorous activity respectively but in no case was the exertion of unusual form.

The almost constant absence of effort in the onset of coronary thrombosis and its connection with rest is significant. The majority of attacks take place at rest or in sleep, especially in the early hours of the morning, when diastolic pressure is lowest and systolic output smallest.

While lowering of diastolic pressure and lessening of cardiac output by reducing coronary flow are important factors in precipitating thrombosis in a person with sclerosed coronary arteries, so in the same person the factors which raise diastolic pressure and increased systolic output lessen the probability of coronary thrombosis. In exertion and excitement these changes take place but with the increasing coronary circulation there is increased demand for it because in persons with coronary sclerosis the heart muscle is still insufficiently supplied with blood. A patient with coronary disease (sclerosis) thus gets

angina with effort and coronary thrombosis while at rest.

The advent of angina does not forebode imminent demise. Most patients live for 5 to 10 years after the onset of the symptoms. Earlier estimates of duration of life averaging 4 to 5 years, reported by Eppinger and Levine (1934), have been revised progressively upwards. Sir Thomas Lewis and Sir James Mackenzie lived and worked for 17 years after their first attack of angina pectoris. Cassidy (1946) reported that coronary disease persisted more than 20 years in 11 of his patients, more than 30 years in 2 of them. The record duration was 52 years in a lady who had her first attack of angina at the age of 30.

VIII. PATHOLOGY OF ARTERIOSCLEROTIC CORONARY ARTERY
OCCCLUSION.

In the majority of cases the pathological basis of cardiac infarction is atheroma of the coronary arteries. Final occlusion is usually due to thrombosis at the site of constriction or atheromatous ulcer. Occasionally cardiac infarction is due to closure of the mouths of the coronary arteries by syphilitic aortitis with or without thrombosis. Coronary embolism is the rarest cause of gross cardiac infarction, and is a complication of infective endocarditis. Polyarteritis nodosa, thrombo-angitis obliterans and acute rheumatic arthritis are other causes.

Mechanism of arterio-sclerotic coronary occlusion:- The process consists of thickening of the intima by cellular hyperplasia and fibrosis associated with the deposition of lipoid and fat. The artery becomes irregularly thickened and may be transformed into a more or less rigid tube. The thickening occurs at the expense of the arterial lumen which may become extremely stenosed.

It was formerly believed that occlusion of an arterio-sclerotic coronary artery was due almost exclusively to obliteration of the lumen by a thrombus deposited on an arterio-sclerotic plaque

or atheromatous ulcer, but the studies of Winternitz (1937), Wartman (1938) and Paterson (1939) have demonstrated that arterio-sclerotic coronary occlusion may occur without thrombosis, and capillary haemorrhages into arterio-sclerotic intima are frequently important in the production of lesion with or without thrombosis. Wartman (1938) found complete occlusion of sclerotic coronary arteries by intermural haemorrhage without thrombosis in six of his cases. Death was attributed to the coronary lesion in all, though in three there was no myocardial infarction.

Horne and Finkelstein (1940) observed that there were 62.5 per cent of closure due to intramural haemorrhage and 37.5 per cent to formation of a thrombus on an arterio-sclerotic plaque in their 100 cases of autopsies of arterio-sclerotic coronary artery occlusion.

The following mechanisms are now believed to occur in the occlusion of an arterio-sclerotic coronary artery:- (a) Intimal haemorrhage with rupture into the lumen and secondary thrombosis: According to Paterson (1939) changes in the coronary arterial pressure, due to hypertension, physical and emotional stresses, may predispose to or precipitate intimal haemorrhage; such haemorrhages occur frequently as part of the arterio-sclerotic

process leading to coronary occlusion. When there is extensive intimal degeneration associated with atheromatous abscess even a small intimal capillary haemorrhage may rupture the arterial endothelium. A thrombus forms at the site of the endothelial tear and leads to the occlusion of the arterial lumen. Occasionally the capillary haemorrhage does not itself rupture the endothelium but may by exaggerating the already extensive intimal degeneration, initiate sub-endothelial and endothelial necrosis and consequent thrombosis.

(b) Intimal haemorrhage with occlusion by intramural haematoma: sometimes the arterio-sclerotic process causes such thickening of the arterial wall that only a very small lumen remains. Rupture of an intimal capillary may result in intramural haematoma. The swelling thus induced may be sufficient to occlude completely the already narrowed arterial lumen even without the occurrence of thrombosis.

(c) Coronary thrombosis on an arterio-sclerotic intimal plaque: In many cases of arterio-sclerotic coronary thrombosis, the occlusion is not precipitated by intimal capillary haemorrhages. The intima may disclose merely dense fibrous thickening and lipoid infiltration or extensive necrosis and atheromatous ulcer. It is probable that the thrombosis precipitated by

arterial endothelial damage by direct impingement of the atheromatous abscess or by rupture of the abscess into the lumen or by other subendothelial changes. Leary (1935) has emphasized the occurrence of coronary artery thrombosis at the site of atheromatous ulcers. Saphir and his associates (1935) in their study of 34 cases stated that recent thrombi of the coronary arteries were found 18 times while organizing and old thrombi were encountered in 14 instances. Occlusions by calcified plaques were observed 13 times. In the 32 instances in which thrombi were encountered in the coronary arteries, the thrombi were located on atheromatous ulcers. In general the thrombosis occurs in the region of the most extensive arterio-sclerotic change and at the point of the greatest narrowing.

(1951)

The recent studies of Leary (1949)/have indicated that athero-sclerosis is a metabolic disease comparable to diabetes and is due to disturbances in cholesterol metabolism. The lesions of the disease are produced focally in the arteries by the deposit of excess of cholesterol in esterform within macrophages at selective sites in the arterial intima.

Early human coronary lesions show the intermittent waves of cholesterophages carrying

excess crystalline ester, cholesterol into the arterial intima. The cholesterol esters stimulate growth of the connective tissue in which the cell lies. The maturing of the connective tissue at the expense of the cholesterophages leads to scarring. Repeated waves of cholesterophages give rise to thickening of coronary intima and narrowing of the lumen.

Considerable research is now in progress in an attempt to find out the part played by blood cholesterol and other lipoids in the genesis of coronary athero-sclerosis. Gertler and Garn (1951) in an investigation of 100 persons who had myocardial infarction prior to the age of 40 years have shown that more significant than the total cholesterol content of the blood which was found to be elevated in most cases was the ratio of such cholesterol to other phospho-lipoids which was considerably higher than the normal. The blood uric acid was also found to be on the higher level and this figure added to the other in form of a quotient has proved of considerable interest.

Location of coronary occlusion.

Autopsy was done on 14 of the 47 cases who died in the ward. Thrombus was found occluding the left coronary artery and its anterior descending branch on three occasions each. Left circumflex artery,

anterior interventricular artery, right coronary artery, circumflex branch of right coronary artery and the main branch of the right coronary artery were each found occluded on one occasion. The occlusion was found about 1 to 2 centimetres from the origin of these vessels. Coronary arteries and their larger branches were severely atheromatous in these cases and the atheroma revealed calcification in many instances.

It was formerly believed that occlusion occurred predominantly in the anterior descending branch of the left coronary artery. Levine and Brown (1929) noted occlusion of the left anterior descending branch 39 times and of the right coronary artery only twice in their 46 fatal cases of coronary thrombosis. More recent studies have disclosed that coronary occlusion occurs with almost equal frequency in the left anterior descending and right circumflex arteries while occlusion of the left circumflex artery develops somewhat less often.

Horne and Finkelstein (1940) in 100 autopsied cases of coronary occlusion observed 61 occlusions of the circumflex branch of the right coronary artery, 56 in the anterior descending branch of the left coronary artery, 27 in the left circumflex, 15 in the (ramus primus) primary branch of the left

descending artery and 7 in the branch to the obtuse margin. The relative freedom from occlusion of the proximal 2 centimetres of the vessel is of particular importance because it permits a free circulation through the first branch or two proximal to the occlusion by means of which a collateral circulation may be maintained.

In this group, in two cases no thrombus was observed macroscopically in any of the coronary arteries or in their larger branches, the arteries were of normal calibre and displayed inconspicuous and patchy atheroma. What are the possible mechanisms by which a sudden decrease in the lumen of an atheromatous artery might be produced? Duguid (1948) believes that atheromatous deposits are the end results of thrombi that are constantly forming up on the intimal coat of the arteries. A major occlusion of a coronary artery may be preceded by attacks of atypical angina of long duration as is well recognised. Dressler (1944) does not consider that these episodes should be distinguished from the more severe attacks producing myocardial infarction and points out that a series of these minor incidents may result in the infarction of a large area of heart muscle. He further states that a raised blood sedimentation rate is frequently found following these minor

episodes which would appear to indicate that permanent changes have occurred in the heart muscles.

Occurrence and Location of Infarction.

In 13 autopsied cases infarction was found in the left ventricle and in the remaining one it was found in both the left and right ventricles. Cardiac infarction is confined almost exclusively to some portion of the left ventricle or the inter-ventricular septum. The right ventricle is involved rarely. Saphir and his associates (1935) observed one instance of right ventricular infarction in their 34 cases of myocardial infarction. Wartman and Hellerstein (1948) found that the left ventricle was the most frequent site of an infarct, being involved in 152 of 160 cases. In the 8 cases in which the left ventricle was not involved infarcts were present in the right atrium and right ventricle in 4 cases each. Whether this is due to a richer coronary anastomosis to the relatively smaller myocardial mass of the right ventricle or to other factors is not known .

Size of the Heart after Cardiac Infarction.

Cardiac enlargement was found in 17 patients with normal or low blood pressure of whom 10 died. The cardiac enlargement was determined by X-ray, electrocardiogram, clinical examination, and in

some cases have been met with at the autopsy. Cardiac dilatation and hypertrophy may develop in patients with coronary artery sclerosis in the absence of hypertension or valvular disease. It is probably a compensatory reaction explained by Starling's law of the heart. Such cardiac enlargement may develop without manifestation of gross heart failure.

Cardiac hypertrophy undoubtedly occurs in coronary artery occlusion in the absence of hypertension, rheumatic cardio-vascular disease or syphilitic aortic insufficiency. In such cases the hypertrophy has been interpreted as direct consequence of ischaemia due to the coronary artery occlusion. Smith, Miller and Graber (1926) observed the development of cardiac hypertrophy following experimental ligation of the coronary artery in dogs.

Cardiac enlargement occurs in at least two thirds of cardiac infarction due to coronary thrombosis. Bartels and Smith (1932) noted cardiac hypertrophy in 37 of 42 cases of cardiac infarction. In their material of study these authors excluded all cases in which there was any condition that is, or is supposed to produce cardiac hypertrophy and thus from the data at their disposal they concluded that cardiac infarction was a definite cause of

cardiac hypertrophy.

According to many writers the cardiac hypertrophy is either due to present or pre-existent hypertension or to congestive heart failure. In the presence of congestive heart failure, marked cardiac hypertrophy was noted whether or not there was associated hypertension.

Palmer (1937) followed 200 cases of coronary thrombosis for an average period of three years. Cardiac enlargement was found in 128 cases while there was no increase in the size of the heart in 72 cases. Hypertension was held to be the single or predominant cause in more than 80 per cent of all cases with enlargement. 4 cases in this group had enlargement apparently due to myocardial ischaemia. Davis and Blumgart (1937) noted the relationship of congestive heart failure to the occurrence of cardiac hypertrophy in a study of 17 cases of heart failure not due to hypertension, valvular disease or other accepted causes of hypertrophy.

Although cardiac hypertrophy is seen pathologically, and cardiac enlargement is observed clinically in many cases of coronary occlusion, the heart is often of normal size in this condition. Hornie and Weiss (1935) reported 20 cases of coronary thrombosis followed roengenotogically from

five months to ten years in all of which the size of the heart was normal. Miller and Weiss (1928) studied 19 hearts at autopsy with old infarctions, none of which disclosed cardiac hypertrophy.

Massie and Miller (1943) studied changes in heart size by the teleoroentgenograms in 16 patients with coronary thrombosis and noted no change in the size or the shape of the heart in 8 of these cases. In the first two weeks following the acute attack an increase in heart size was observed in only 2 cases of the entire series.

IX. SYMPTOMATOLOGY.PRE-MONITARY PAIN.

It has been recognised since the earliest clinical studies of coronary thrombosis that while the illness often commences without warning there are instances in which pre-monitory pains foreshadow the event. During the analysis of this series of cases it became evident that attacks of cardiac pain of relatively short duration, not uncommonly occurred for the first time in days or weeks immediately before the coronary occlusion. Furthermore in most cases the features of these attacks differed considerably from those of typical angina pectoris. Premonitory pain was found in 37 cases in this series.

Wearn (1923) wrote "that the onset may occur after a number of anginal attacks, which together with dyspnoea may constitute the only previous warnings of involvement of coronary arteries." Parkinson and Bedford (1928) and Levine (1929) drew attention to the same fact. The published references to premonitory pain since then have been numerous and include those of Willius (1936), Feil (1937), Sampson and Eliaser (1937), Brill (1938) and Yater et al (1948). Behrmann et al (1950) have described the following modes of onset of

cardiac infarction:- (1) Painless infarction, (2) intermittent pains without severe prolonged pain, (3) sudden onset of myocardial infarction without any preceding pain, (4) premonitory pain of short duration followed by prolonged infarction pain and (5) preceding angina pectoris of some duration with or without premonitory pain. These authors have indicated that premonitory pain was most likely due to rapidly forming thrombus, or subintimal haemorrhage with a fairly rapid occlusion of a coronary artery or its major branch. Mounsey (1951) has recently laid down the criteria for acceptance of symptoms as "prodromal" and has detailed the clinical and cardiographic feature of this state. His findings support the concept of a gradually decreasing coronary lumen with increasing myocardial ischaemia as the cause of the pain terminating in infarction. He believes that the fatality rate is lower among patients who show prodromal symptoms than among those who do not show them.

PAIN.

Pain is a classical sign of coronary thrombosis. It was found in 117 patients, while in 11 there was no pain but only a tightness behind the sternum, sub-sternal discomfort and breathlessness. In 63 patients the pain started as angina of effort and as the time went on it increased in frequency, developing on slight exertion and not to be relieved on resting or by nitroglycerin tablets. In the remaining 54 the pain had been acute, more crushing in intensity, and intolerable and prolonged in duration. It was described by the patients as squeezing, constricting, choking, tearing, dull and boring and sometimes burning in quality. In 40 cases the pain was accompanied by breathlessness and in 23 nausea and vomiting was present, while marked restlessness was found in 21.

Location and radiation of pain. The pain was localised mostly to the retrosternal region. It often spread to both sides of the chest especially to the left. There was no radiation of pain in 35 patients of whom 13 died. It radiated to the left arm in 30 patients of whom 8 died, right arm in 6 patients of whom 2 died and to both arms in 24 patients of whom 11 died. It also radiated to the right shoulder 4 times, into the side of the neck 15 times, through the back 9 times,

between the shoulders 4 times and into the jaws and gums 3 times. Epigastric pain was found in 11 patients. The pain persisted in varying degrees for many hours. Mortality was higher in the group where the pain radiated to both the arms.

The similarities in quality, location and radiation of the pain of coronary thrombosis like that of angina pectoris is due to myocardial ischaemia. In angina pectoris the pain is brief and paroxysmal as the myocardial ischaemia is produced by effort or emotional states, which are themselves transient. The pain subsides with the cessation of the causative factor or the administration of a vasodilator like nitroglycerin, because the myocardial anoxia is reversible and there is no significant permanent anatomic change. In the case of coronary thrombosis the pain is prolonged because myocardial anoxia is usually due to mechanical occlusion of a coronary artery which is not transient or reversible; the artery is not capable of dilatation by nitrites. The greater severity of pain of coronary thrombosis as compared to that of angina pectoris is probably an expression of more extensive myocardial anoxia and of longer duration.

Nausea and vomiting was usually associated with pain and shock. In some cases it appeared

after the patient had received a morphine injection. In most of the cases it seemed to be a reflex phenomena. Restlessness of all degrees, from moderate uneasiness to profound agitation was found. Unlike angina pectoris a patient with painful infarction soon finds out that activity or posture is powerless to relieve pain. Dyspnoea was the most frequent symptom in the acute cases. The sensation of difficulty in breathing early in an attack was often associated with a feeling of constriction.

SILENT OR ATYPICAL CASES OF MYOCARDIAL INFARCTION.

Pain was not experienced by 11 patients. In these cases there was only tightness behind the sternum, substernal discomfort and breathlessness. Dyspnoea was a marked feature and 6 of the 11 died. Weakness, cold sweat and collapse was also found in these patients. Mortality was higher in this group as compared to the pain group. Davis (1932) analyzed 53 autopsied cases of coronary thrombosis and found no history of pain in 21 of them. Pain is said to be uncommon in negroes, in whom breathlessness is the usual clinical manifestation.

Gorham and Martin (1938) in a study of 100 necropsy cases of myocardial infarction, reported that 42 had experienced no pain. These patients belonged to the older age group and few of them

were hypertensive and had suffered previous effort pain. In their series there were 29 cases of old healed infarcts and it was in this group that a history of pain was most commonly absent. 12 of the painless cases were in a group of 17 showing fibrotic narrowing of the coronary arteries without actual thrombosis, and old infarction without pericarditis, Kennedy (1937) considers that the frequency of painless infarctions had been overstressed. In a study of 152 cases he reported that in 91 per cent of recent and in 64 per cent of old infarctions there was classical coronary pain. Pollard (1940) in a study of 375 cases of myocardial infarction, found 17 instances in which no pain, substernal pressure or other anginal symptoms had occurred at any time. In these cases the most common symptoms were dyspnoea, nausea and vomiting, dizziness and fainting or collapse. In his series there were 15 additional cases in which there had been no pain or anginal symptoms and in which the presence of myocardial infarction was strongly suspected even though the electrocardiographic findings were not pathognomic. These authors believe that painless coronary occlusion is relatively uncommon.

SHOCK.

It was found in varying degree at the time of admission. In 38 patients shock was marked, in 52 it was mild, while a feeling of exhaustion and weakness was experienced by 10 patients and in the rest there was no shock. A severe degree of shock was associated with higher mortality.

Shock usually occurs soon after the onset of myocardial infarction and lasts a few hours to one or two days. In fatal cases it may persist longer until the patient succumbs. Shock in acute myocardial infarction is probably due to the sharp and sudden reduction of cardiac output resulting from myocardial injury.

X. OBJECTIVE MANIFESTATION OF ACUTE MYOCARDIAL
INFARCTION.

PERICARDIAL RUB.

Pericardial friction rub was found in ten patients in this Series. Of these four had anterior infarct, two, posterior infarct, one antero posterior and in three there was no electro-cardiographic evidence of the infarction. Six of these patients died in the wards. The friction rub was found intermittently. It usually developed on the 2nd or 3rd day of the attack and was audible for a few days.

Occasionally with large infarctions or progressive myocardial infarction a pericardial rub persists for many days. A pericardial friction rub is not always indicative of an anterior infarction as is commonly believed. It may be heard also following posterior infarction. It is much more likely to appear when there is a generalised pericarditis than with a localised pericarditis and generalised pericarditis may follow posterior as well as anterior wall infarction.

The pericardial friction rub has been noted more frequently. It is likely that in some cases the condition may have been missed on auscultation because the rub is transitory and ordinarily not

loud. In the Series of Bean (1938) pericarditis was found at autopsy 58 times and a rub had been heard in 24 cases or 41 per cent.

When present in a patient suspected of having coronary thrombosis, a localised pericarditis is of great diagnostic value.

BLOOD PRESSURE AND PULSE RATE.

Blood pressure persistently below 100 mm. Hg was found in 25 patients, of which 16 died. The immediate mortality in this group was 64 per cent, compared to an overall mortality of 36.7 per cent. Similarly the pulse rate was also analysed and it was found that 36 patients had a persistent pulse rate over 100 beats per minute. 24 of these patients died within six weeks. The mortality rate in this group was 66.6 per cent. Mortality rate was considerably increased when either the systolic pressure fell below 100 mm. Hg or the pulse rate rose to over 100. It is interesting to note that the majority of cases in whom death took place in the ward showed both persistent low blood pressure, i.e. below 100 mm. Hg, and persistent fast pulse, i.e. above 100 per minute.

The pulse rate is usually accelerated between 100 and 110 beats per minute, especially during the

febrile stage. The rhythm of the pulse is usually regular but premature beats occur commonly and occasionally. The blood pressure as a rule is followed by a fall in cardiac infarction. The fall in blood pressure usually occurs rapidly, sometimes after several hours, but more often on the second day of the attack. The fall in blood pressure after the acute myocardial infarction is probably due to the reduction in cardiac output. The blood pressure remains normal or returns to normal in patients who survive an attack of acute coronary thrombosis.

FEVER.

The temperature was taken in the mouth and fever was observed in 71 per cent of the patients. It was generally found from the beginning or within twenty four to forty eight hours from the onset. The rise in temperature was usually moderate, being 99 to 101 degrees Fahrenheit and reaching 102° F. in a few, lasting 2 to 4 days in uncomplicated cases. In general the larger infarcts gave rise to higher and more sustained fever. Terminal rise in temperature was usually found in patients dying primarily of congestive failure, infarcts of the lungs or pneumonia.

It was found that fever and leucocytosis appeared together in 58 cases, leucocytosis without

fever in 13 cases and fever alone in 15 cases. (N.B. white Blood cell count was recorded in 78 patients only). It may be stated that infarcted tissue in the body probably liberates toxic products that produces leucocytosis and fever, moreover congestive heart failure alone can produce fever.

Levine (1929) has emphasized the fact that oral temperature determination is not reliable. Many cases with no fever had a large degree of shock and dyspnoea which interfered with satisfactory observation of mouth temperature.

LEUCOCYTOSIS.

The white blood cell count was recorded in 78 patients. A white blood cell count of 6000-9000 per c.mm. was found in 32 patients of whom 2 died, 9000 to 12000 per c.mm. in 21 patients of whom 5 died, 12000-15000 per c.mm. in 10 patients of whom 7 died and 15000 per c.mm. and above in 5 patients of whom 3 died. The highest leucocyte count was 16800 per c.mm. The mortality rate appears to increase with the rise in leucocytosis; in fact marked leucocytosis indicated a poor immediate outlook. With a few exceptions leucocytosis appeared on the second and third day.

Table XII. Showing leucocytosis and mortality.

White blood cells per c.c.	No. of patients	Mortality	Percentage
6000-9000	32	2	6.25
9000-12000	21	5	23.8
12000-15000	10	7	70
15000+	5	3	60

Libman and Sacks (1927) stated that leucocytosis may develop as early as one and a quarter hours after the onset of the symptoms. In the series of Parkinson and Bedford (1928) the leucocytosis from 10,000 to 15,000 was found commonly but not invariably, the highest leucocyte count was 25,800. In the series of Bellings et al (1949) 12 patients had white blood cell count of more than 25,000 c.mm. 9 died within 30 days. Similarly Chambers (1946) reported that with white blood cell counts between 20,000 and 25,000 the prognosis was grave but with those above 25,000 the mortality was 100 per cent.

BLOOD SEDIMENTATION RATE.

Blood sedimentation rate of 20-29 mm. per hour was found in 13 patients of whom three died, 30-39 mm. per hour in 12 patients of whom 5 died, 40-49 mm. per hour in 4 patients of whom 2 died, and 50 mm. per hour and above in 5 patients of whom 3 died. The highest sedimentation rate was 105 mm. per hour and was found in one patient only who

died.

Table XIII Showing increased sedimentation rate and mortality.

Blood sedimentation rate per hour	No. of patients	Mortality	Percentage
20-29	13	3	23
30-39	12	5	41.6
40-49	4	2	50
50+	5	3	60

The rate of sedimentation of erythrocytes is almost always increased following an acute myocardial infarction. The increased sedimentation rate is usually first noted on the 2nd or 3rd day of the episode and persists for several weeks until the infarct is healed. The increased sedimentation rate is attributed to a change in the composition of the plasma due to the absorption of products of the necrotic heart muscle.

Other Symptoms encountered in the series were:- auricular fibrillation in five patients, extra-cystoles in three patients and palpitation in four patients. Orthopnoea was experienced by five patients, while Gallop rhythm, Cheyne-Stokes breathing, was occasionally found in a few. The heart sounds were distant i.e. tic-tac. Oedema of the lungs was met with in three patients.

XI. ELECTROCARDIOGRAPHIC DIAGNOSIS.

There were 49 anterior type of infarct, 33 posterior type of infarct. 17 patients died before any electrocardiograph was taken. 21 patients had an infarct which could not be localised on electrocardiographic evidence. Of these patients 14.3 per cent died in the wards.

Electrocardiographic signs characteristic of infarction are not always found. When the clinical picture obviously indicates this diagnosis, the electrocardiogram may even present a normal outline. The latter occurs when the infarct is small, when it is entirely intramural, i.e. does not reach the endocardial or epicardial surfaces, when it is unfavourably situated in regard to the leads obtained, and when a left bundle block is present. Occasionally the appearance of the electrocardiographic signs of acute infarction is delayed some days until the infarct becomes larger by extension. In some cases changes in Q.R's complex are noted after the occurrence of anginal pain and although these changes are not of a characteristic kind, infarction cannot be excluded.

Gilchrist and Ritchie (1930) pointed out that while rapid changes in the ventricular complex in serial electrocardiograms are strong presumptive evidence of myocardial infarction, similar changes developing over a long period of time may be due

to progressive myocardial fibrosis following coronary sclerosis.

In this series the anterior infarcts were more than the posterior and the other. Mortality was 62 per cent in the antero-posterior type, 33 per cent in the anterior type and 16 per cent in the posterior type. The posterior type of infarction seems less fatal.

Table XIV. Showing type of infarction and mortality.

<u>Type of infarction</u>	<u>Number of cases</u>	<u>deaths</u>	<u>percentage</u>
Anterior	49	16	33%
Posterior	33	5	16%
Antero-posterior	8	5	62%

The anterior type of infarctions are more common. This is perhaps due to the descending branch of the left coronary artery usually being the vessel occluded. Some structural defect or other abnormality in the artery or the increased bulk of the myocardium of the left ventricle, as compared with the right ventricle, may explain the predominance of anterior infarction.

Wood and his associates (1933) believe that anterior infarction is far more serious than the variety involving the posterior wall. Barnes and Ball (1932) say that the mortality rate is the same in both anterior and posterior varieties.

In his series of 100 cases, Chambers (1946) found 52 anterior-apex type (QT1) 40, the posterior-base type (QT3) and 8 were a combination of both types. The mortality was higher in the anterior-apex type, which was 47 per cent, than in the posterior-base type, 38 per cent. In patients with a combination of both types the mortality was 100 per cent.

Levine and Rosenbaum (1941) noted that anterior infarct was more frequent in the initial attack than later. In Baer and Frankel's (1944) 52 per cent of the cases were of the anterior type and 34 per cent of the posterior type. Rathe (1942) observed a definite predominance of anterior and intermediate electrocardiographic patterns in early fatal cases. Willius (1936) and Master et al (1936) attached little prognostic significance to the localisation of the infarct.

XII. COMPLICATIONS.CONGESTIVE HEART FAILURE.

In this series there were 19 instances of congestive heart failure, both left sided and combined failure. There were 14 males and 5 females. The average age of males was 63.57 years and the average age for females was 69.6 years. Of the 14 men 9 died and of the 5 women 2 died.

Congestive heart failure is an extremely grave sign in both men and women but especially so in men. This is obvious, since the ordinary active older man has greater level of activity than the ordinary older woman. Rest in a man therefore is a greater departure from ordinary activity. Hence when failure occurs when he is at rest it might be more ominous. Myocardial infarction may exhibit signs and symptoms of congestive heart failure at the onset of the attack or during the first few days.

Master and his associates (1937) observed that congestive heart failure occurred in 66% of patients with acute coronary thrombosis in their Series. It usually consisted of failure of both ventricles, occasionally of the left ventricle alone. Reports have also appeared of the occasional occurrence of acute pulmonary oedema or

of sudden aggravation of pre-existing heart failure as the only indication of myocardial infarction. In such patients there is usually to be found some form of pre-existing cardiac damage due to hypertension, previous myocardial infarction or coronary sclerosis. Myocardial infarction involving a large area of the heart has been reported to be invariably accompanied with congestive heart failure. The incidence of congestive heart failure is found to increase with the age. Master and his associates (1937) found that congestive heart failure occurred with the same frequency in both sexes.

Gross and Engelberg (1940) reported a high incidence of congestive heart failure in their Series. 90 of 100 cases were showing congestive heart failure.

Master and his associates (1937) observed that many patients with congestive cardiac failure associated with recent myocardial infarction are also in Shock and that in fatal cases the congestive failure usually predominates except when death occurs early, in the later instance Shock predominates. All the observers agree that congestive heart failure is of grave prognostic significance in patients with myocardial infarction.

THROMBO-EMBOLIC PHENOMENA (EMBOLIZATION).

There were five instances of pulmonary and cerebral emboli, of these 3 patients died. Pulmonary emboli may be only in part a direct consequence of the cardiac infarction and slowing of the peripheral circulation; enforced rest in bed, with lack of movement of the extremities and the associated venous stasis are probably more important causes. These factors contribute to the formation of phlebo-thrombosis of the extremities which in turn is the common cause of pulmonary emboli. Cerebral emboli, like other emboli to the Systemic arteries originate in left ventricular mural thrombi. The frequency and importance of thrombo-embolic lesions in acute myocardial infarction has gained increasing significance recently because of the possibility of treatment with anti coagulants and surgery.

Thrombo-embolic phenomena have been recorded by many observers.

Parkinson and Bedford (1928) emphasized that involvement of the intra ventricular septum in myocardial infarction may cause thrombosis of both ventricles leading to pulmonary and systemic emboli. Nay and Barnes (1945) observed that the emboli arising from the left side of the heart are found most often in the spleen, kidney and brain and less

commonly in the mesenteric vessels and the extremities. Rosenbaum and Levine (1941) stated that most pulmonary emboli arise from veins of pelvis and the extremities and that pulmonary occlusion to a lesser extent may arise from local thrombosis of pulmonary vessels. Levine and Brown (1929) reported that many instances of hemiplegia are due to emboli arising from the heart following acute myocardial infarction. Dozzi (1937) analysed 1000 consecutive necropsies and found that 29 per cent of patients with myocardial infarction had cerebral thrombosis or embolism. Askey and Neurath (1945) stressed the fact that emboli may arise in the heart during auricular fibrillation. Garvin (1942) in a series of 771 autopsies found that in about 60 per cent of the cases of myocardial infarction there were one or more infarcts in lungs, brain, kidneys, spleen, extremities and intestines. In a review of cases reported in the literature, Hellerstein and Martin (1947) found an average of 11.5 per cent of embolism or infarction. In their own autopsy Series of 160 cases 73 or 45% were associated with embolism, which was the main cause of death in 12% and a contributory cause in an additional 15 per cent. All observers agree that emboli and thrombotic phenomena were of grave prognostic significance in patients with myocardial infarction.

SUDDEN DEATH.

47 patients in all died in the wards within six weeks. Of these 19 patients died within 24 hrs. 10 patients who were progressing satisfactorily for varying times i.e. 10 to 21 days died suddenly. They were found in a state of collapse one day and died within minutes after a few gasping breaths. 6 of these deaths were probably due to an extension of the previous episode. In the remaining 4 emboli of lungs and brain were found in 3 at autopsy and in the 4th autopsy revealed a small rupture (vent) in the posterior wall of the left ventricle. Applebaum and Nicolson (1935) reported rupture of the heart in 9 out of 150 cases of coronary occlusion. Rupture occurred through the left ventricle in all cases.

Sudden death is a common termination of acute myocardial infarction. It may be the only manifestation of the attack or it may occur in any stage of the acute disease during convalescence. It is believed that most often sudden death results from ventricular fibrillation. In some instances sudden death may result from cardiac standstill due to vagovagal reflexes from the infarcted heart muscles.

Falk (1942) discussing the causes of sudden

death after coronary occlusion states that ventricular fibrillation has been reported by several observers who took electrocardiograms before death. Massive pulmonary embolism is another immediate cause of death which occurs following coronary occlusion. The emboli do not come from the heart but usually from the iliac veins. Thrombosis of the iliac veins is favoured by the lowered blood pressure and prolonged bed rest. Myocardial failure with pulmonary oedema has been noted during recovery from coronary occlusion. Death is not an infrequent result of this complication.

XIII. MORTALITY.

In this series 97 cases of thrombosis occurred in males and 31 in females. 33 males (34%) and 14 females (45%) died in the ward within six weeks of the onset of acute coronary thrombosis. 19 cases died during the first 24 hours of admission. 14 patients had a previous history of coronary thrombosis. Mortality in this group was 55 per cent. Three patients had their third attack and two of them died. One patient had his first attack of coronary thrombosis in 1928. The shortest duration was 5 months. 6 patients had their further extension of fresh episodes while in the ward. All 6 died within the immediate period, i.e. 6 weeks. In persons of fifty years or older there were more deaths within six weeks of the acute episode than among those under 50. The average age at the time of death was 62 years for males and 65.5 years for females. In the series of Mintz and Katz (1947) the average age of the females at the time of death was 63.4 years and the males 59.6 years. The immediate mortality rate was higher in females than in males. The relatively large number of females over fifty years of age was probably a contributing factor. Moreover it may also be explained by the fact that

the initial attack occurred later in women than in men and the immediate mortality was increased with advancing years.

The effect of age on immediate mortality has been pointed out by many writers. Cooksey (1935) writes that in his series of 53 cases of coronary thrombosis he found the mortality at the age of 60 and above distinctly higher than at ages below 60. Rathe (1942) reports that early fatal cases in his series were 3 years older than the survival group and the females were older than the males. The immediate mortality rate for men in the series of Woods (1941) was 41.7% as compared with 75% for women. The immediate mortality rate was 57% for men more than 60 years of age and 84.6 per cent for women in the same age group. His study showed that the immediate mortality of acute coronary thrombosis increases steadily with advancing age. Baer (1944) considers that (1) the younger individual has a somewhat better immediate prognosis, (2) the mortality is slightly higher in women, and (3) the mortality increases with advancing years. It has often been contended by students of this subject that individuals above 60 years of age tolerate acute coronary occlusions better than those below 60 years of age. This study does not bear out such a contention and would seem much more in

harmony with clinical experience in which other circulatory insult is tolerated less well by such elderly patients.

The mortality figure (36.7%) is higher than 16.2% found by Conner and Holt (1930) and 16.5 per cent cited by Master et al (1936). However all the patients were ill enough to require admission to a hospital and thus the series does not include those with a milder form of illness who did not come to a physician until after they had recovered or who could be cared for at home. The mortality in this series is definitely lower than 53 per cent reported by Levine and Brown (1929), 38.6 per cent of Cooksey (1935) and 40.4 per cent found by Bellings et al (1949).

Table XV.

Age and sex incidence and immediate mortality (death in six weeks)

<u>Age in years</u>	<u>Total</u>	<u>Male</u>	<u>Female</u>
	No. of Immediate patients mortality	No. of Immediate patients mortality	No. of Immediate patients mortality
40-49	23	20	3
50-59	36	30	6
60-69	46	33	13
70-79	22	13	9
80+	1	1	-
	128	97	31
	47	33	14

Reports of immediate mortality in the literature vary widely from 53 per cent of Levine and Brown (1929) to the 16.2 per cent of Conner and Holt (1930). Many of the authors were only considering the first attack in which the prognosis is better than after subsequent attacks. Records of some of the series are given below:-

Table XVI.

Immediate mortality rate after myocardial infarction.

<u>Authors</u>	<u>No. of cases</u>	<u>Mortality rate</u>
1. Levine and Brown (1929)	145	53%
2. Conner and Holt (1930)	284	16.2%
3. Cooksey (1935)	53	39.6%
4. Master et al (1936)	267	16.5%
5. Master et al (1937)	140	21%
6. Bland and White (1941)	200	19%
7. Levine and Rosenbaum (1941)	208	33%
8. Woods and Barnes (1941)	128	46.9%
9. Rathe (1942)	274	20%
10. Newman (1946)	50	78%
11. Katz and Mintz (1947)	572	21.8%
12. Yater et al (1948)	866	50.1%
13. Bellings et al (1949)	240	40.4%
14. Present Series	128	36.7%.

In the series of Bland and White, Yarter et al and Bellings et al the mortality rate is calculated over the first four weeks and 30 days.

In the series of Newman and Yater the great majority occurred within 24 hours. 33 of Newman's 39 fatal cases were found dead or died almost immediately after a collapse. In 375 of Yater's 434 cases death took place in the first 24 hours.

Death occurs most commonly in the first day, being either sudden or rapid in onset or in the first few days when there is commonly a persistent severe degree of peripheral circulatory failure. Master (1935) says that if the patient lives through the first 24 hours, the chances are he will survive that particular attack. In the subsequent weeks the death may be due to heart failure, to the recurrence of the infarct or some other major thrombo-embolic complication. In a study of 140 cases Master (1937) reported that the mortality rate was 30 per cent in the presence of heart failure and only 4 per cent when it was absent.

The factors influencing the mortality are:-

1. Age.
2. Number of previous infarcts.
3. State of the individual at the time of onset.

All have a bearing on the adequacy of the circulation to the remaining myocardium and on the efficiency of that myocardium.

XIV. TREATMENT.

The treatment of coronary thrombosis is by no means standardised, nor do all physicians who are confronted with the problem agree on the effectiveness of the various medications used. The treatment of each case of coronary occlusion must be individualized.

The treatment is designed (1) to reduce the work of the heart until the infarcted area is healed, (2) to alleviate pain or other discomfort, (3) to overcome shock and cardiac failure if present, and (4) to cope with dangerous cardiac arrhythmias or any complications that may arise.

The patients in this series were treated with anti-coagulant therapy and routine treatment in hospital, which consisted of (1) rest in bed in the most comfortable position, usually propped up with two pillows, (2) no activity for 3 weeks and then a gradual return to it, (3) morphine for the relief of pain, (4) sedation with phenobarbitone during the day and Nembutal at night, (5) light diet, (6) attention to the bowels with enema after 4 days if required, (7) graduated exercises for the limbs and breathing exercises under a physio-therapist from the beginning of the 4th week, and, (8) treatment of the complications as they arose - digitalis, mercurial, diuretics, oxygen and morphine

as required for heart failure, Aminophylline 0.25 gm. I.V. followed by cardiophylline 0.1 gm three times a day by mouth was given for 3 weeks as a routine. At the end of the 5th week or during the 6th week the patient was allowed up and after a few days discharged home.

REST.

Rest in bed is the mainstay of the treatment, generally for an average period of six weeks. The rest period is based on the belief that six weeks are required for the infarct to be converted into a firm scar. Most of the fatalities and complications from myocardial infarction occur during the first week, a more moderate number in the 2nd week and relatively few in the third week. The average period of six weeks is designed for recovery from the acute attack and also for healing of the infarction. The rest in bed may not only mean avoidance of physical exercises but complete mental repose and protection from emotional strain. The important advantages ascribed to bed rest are avoidance of cerebral anoxia, during shock, prevention of sudden death and preservation of myocardial function.

The rigid severity with which bed rest is generally enforced has recently been debated as disadvantages of recumbancy are many. Circulation

to the bases of lungs is poor, atelectasis, hypostatic pneumonia and constipation are common. Incidence of phlebo-thrombosis and subsequent embolization is much higher. Other disadvantages include abnormal nitrogen and calcium metabolism and deterioration in mechanisms essential for adequate circulation in the erect position. Finally the psycho-genic sequelae are important, with alarming frequency patients who have had myocardial infarction are seen to have been pitifully inactive because of symptoms brought on by fear rather than by heart disease. There is thus a growing tendency to treat cases of coronary thrombosis out of bed as soon as the significant symptoms of shock and pain has subsided. Collateral circulation develops only by demand of insufficient blood supply. Possibly increasing activity during part of the healing phase would tend to produce a collateral circulation more adequate for normal function. Decreasing the period of bed rest also tends to lessen the incidence of pneumonia and the troublesome complication of painful joints.

Levine (1951) has treated 70 cases of acute coronary thrombosis in a chair in addition to other therapeutic measures for varying and increasing periods of the day, beginning not later

than the first week of the attack and there were only 7 deaths. The immediate mortality of about 10 per cent is low enough to encourage the belief that this method of treatment does not carry any risk with it. The author believes that the treatment has both physical and psychic advantages over the strict bed rest method of treatment.

MORPHINE.

Morphine is administered to control severe pain, restlessness and anxiety at the onset of the attack but it is not essential if the pain is relatively mild and can be relieved by weaker analgesics. Morphine is the most valuable drug of all in the treatment of coronary occlusion but its administration is not without danger and a diagnosis of coronary thrombosis does not in itself justify its use. If pain, restlessness or pulmonary oedema are marked features of the case, then it should be given without hesitation. If these are minimal it may well be advisable to withhold it. It is most important that the patients should obtain an adequate amount of sleep.

OXYGEN.

The administration of oxygen has become an important therapeutic measure. Cyanosis is the clearest clinical indication for oxygen therapy. Barach and Levy (1934) have emphasized its

importance and reported striking relief of pain and restlessness, slower and less laboured respiration, diminution or abolition of cyanosis, disappearance of Cheyne-Stokes respiration within one to three hours of beginning to administer oxygen. These authors consider that at times oxygen may be life saving. It has been used with success in the wards.

Aminophylline has a mild dilator effect upon the coronary arteries in addition to its action as diuretic and appears to be of some value in the treatment of coronary thrombosis.

DIGITALIS.

The position of digitalis in the treatment of coronary infarction has long been in dispute.

Fishberg considers that digitalis does not seem indicated in patients with an initial attack of coronary thrombosis with a competent heart as it carries with it conceivable dangers of:-

- (1) detachment of parietal thrombi with embolization
- (2) rupture of the heart as a result of more forceful contraction, and, (3) ventricular tachycardia or fibrillation as a result of greater irritability of the ventricular muscle.

Askey and Neurath (1946) have also investigated the question of digitalis in the treatment of myocardial infarction. They reported that the risks are those of the production of ventricular

fibrillation by shortening the refractory period of the heart muscle, an increased likelihood of rupture of the myocardium due to the increased strength of contraction, vaso-constriction of coronary arteries by an increase in vagal tone and an increased tendency to thrombo-embolic complications on account of a decrease in the clotting time. In their opinion the likelihood of thrombo-embolic phenomenon is increased by the administration of digitalis, but there is no evidence to show that ventricular fibrillation, myocardial rupture or sudden death occur more frequently after the administration of digitalis. These authors believe that digitalis is particularly dangerous in patients with coronary thrombosis showing signs of congestive heart failure. In their series they found the worst results from digitalis alone in 32 patients with congestive failure of whom 31 died. 13 of these deaths were due to emboli to the systemic circulation. Though these figures are suggestive, they cannot be regarded as final until further controlled experimental studies are carried out.

QUINIDINE.

Routine use of this drug in acute cardiac infarction is advocated by some, on the assumption that by increasing the refractory period of the

heart muscle it diminishes the possibility of ventricular fibrillation. Quinidine is not always a safe remedy and may have toxic effects upon the heart muscle resulting in sudden death. Its use may well safely be reserved for those cases in whom there is persistent auricular flutter and fibrillation and also likelihood of developing ventricular fibrillation.

ANTI-COAGULANTS.

The most important advance in the treatment of coronary thrombosis has been the introduction of anti-coagulants. This form of treatment was suggested by Best in 1938. It is now a well established and generally recognised fact that the injection of heparin into the experimental animal or into man is followed shortly by a prolongation of the coagulation time of the whole blood. It has been shown that myocardial infarction with thrombus formation in coronary arteries and endocardial mural thrombus formation could be prevented by the administration of heparin.

Anticoagulant therapy has greatly reduced the risk of thrombo-embolic complications. Wright et al (1948) treated 432 patients with anti-coagulant therapy in addition to conventional therapy and 368 patients in the control group who received only conventional therapy. Thrombo-embolic

complications occurred in 25 per cent of the controls and in 11 per cent of the anti-coagulant treated patients. Gilbert and associates (1949) consider that the decrease in thrombo-embolic phenomena is due to the increase of the myocardial blood supply effected by the coronary vaso-dilator drugs. As a result of increased blood supply there is less damage of the endocardium and hence there is less tendency to the formation of mural thrombi. These authors believe that anti-coagulant drugs, heparin and dicumarol have such a coronary vaso-dilator effect.

DIET.

The major change in the dietetic treatment of coronary thrombosis was made in 1935 when Master introduced his low calorie diet. It consists of 750-850, usually 800 calories. It is a well balanced diet and contains approximately 80 grams of carbohydrates, 50 grams of protein and 30 grams of fat, with vitamins and adequate calcium. The diet is given to the patient until he is ready to leave his bed. The fluid intake is limited from 1000 cc to 1200 cc. The patient loses weight while on this diet and there is a fall in the basal metabolic rate to -20 and -30 per cent. Clinically the patients lose their pain more quickly and are less apt to have gastro-intestinal

complaints. Their vital capacity, blood velocity, venous pressure and blood pressure return more quickly to normal. Master (1935) treated 75 patients in 85 attacks of coronary thrombosis by a regime of low (800) calorie diet. 8 patients died - only one in a first attack. Such a strict diet was not however used in this series but the diet has been light and about 1200-1500 calories given.

XV. FOLLOW UP STUDY.

In the follow up study of the cases from 6 months to 4 years, 50 patients were traced. This group consisted of 16 patients with antecedent angina and 34 without a previous history of angina. 10 of the 50 patients had died. In the fatal group there were 3 patients with an antecedent angina pectoris and the rest with no previous history of angina pectoris, the mortality in the anginal group was 18.7% and in the non-anginal group 20.6%. The mortality thus was relatively higher in those who had no antecedent angina pectoris, than those in whom the coronary episode had followed angina pectoris.

4 patients died from 6 months to 1 year from the onset of coronary thrombosis, 5 died from 1 year to 1 year and 10 months and 1 died within 3 months. Three patients^{have}/lived more than 3 years i.e. 40 months, 42 months and 47 months, and are still alive.

12 patients had returned to their original work and 4 had taken up lighter jobs. 12 patients had retired and 14 were doing no work.

Shortness of breath was complained of by 17 patients while 23 did not complain of any such symptom. The shortness of breath denotes the condition of coronary arteries, one may surmise as

to what part cardiac decompensation might play in this.

Pain in chest on exertion or at rest was experienced by 19 patients while 21 did not have it.

Swelling of feet and ankles was stated by five patients and 9 patients declared their inability to sleep flat in the bed at night. 11 patients suffered from fast beating heart.

The general health of patients as judged by the above investigation was good in 23 cases, fair in 10 cases and poor in 7 cases.

XVI. SUMMARY AND CONCLUSION.

1. A historical review of coronary thrombosis is presented.
2. The increasing incidence of coronary thrombosis has been investigated by a study of the annual reports of the Registrar General for Scotland between 1939 and 1949 and data from other sources. The number of deaths certified as due to coronary thrombosis has increased yearly between 1939 and 1949. The increase has been more striking among females than males. One of the disconcerting facts regarding the present situation is the increasing incidence of coronary thrombosis among younger persons.
3. A Study of 128 patients with acute coronary thrombosis was made. These patients were treated in wards 23-24 Royal Infirmary Edinburgh from 1st January 1948 to December 1951. The purpose of the study was to determine the relationship of coronary thrombosis to angina pectoris.
4. Mortality:- The immediate mortality in the entire series was 36.7 per cent. The average age at the time of death was 62 years for males and 65.5 for females. 14 patients

had a previous history of coronary thrombosis. Mortality in this group was 55 per cent. Mortality was higher in persons of fifty years or older than those under fifty. The immediate mortality rate was also higher in women than in men. The relatively large number of females over fifty years of age was probably a contributing factor. Mortality was increased with advancing years.

The factors influencing the mortality are:-

1. Age. 2. Number of previous infarcts.
3. The State of the individual at the time of onset.
5. Electro-cardiographic findings:- These revealed that the anterior infarctions were more common than the posterior and the antero-posterior. The ratio between the anterior and posterior infarcts being about 7:5. Mortality was greater in the anterior type (33%) and greatest in antero-post type (62%). The group with no electro-cardiographic evidence of infarction had a lower mortality.
6. Sex:- The sex ratio was 3.1 males to 1 female and prognosis for women somewhat grave. More males than females are prone to develop coronary thrombosis, the reason for this is

that the intima of the coronary arteries is much thicker in males than females and the predilection of atherosclerosis for the coronary arteries is due to their possessing an intima varying in thickness from 10 to 600 per cent of that of the media and averaging 26 per cent in new born males and 8 per cent in females.

7. Age:- The average age at the time of attack was 60 years for the entire Series. The youngest patient was aged 42 years and the oldest 81 years. 23 patients were under 50 years of age and 105 over 50 years. The average age at the onset was 58.5 years for males and 64 years for females. In most males the attack occurred in the 6th and 7th decades whereas in females the onset was greatest in the 7th decade. Athero-sclerosis perhaps develops later in women than in men.
8. Hypertension:- Antecedent hypertension was found in 50.4 per cent of patients. 41% of men and 64.5% of women were hypertensive. The immediate mortality following coronary thrombosis was lower (35%) than that of the non-hypertensive group (38%). Coronary occlusion in women who had neither previous angina pectoris nor hypertension was relatively rare.

9. Diabetes mellitus:- Only four cases were found in the whole Series.
10. Occupation:- No particular inference could be drawn from the data. The disease is found in all shades of life and is not particularly prevalent in any class or occupation.
11. Race:- All the patients in the Series belonged to Caucasian race, therefore no comparison with other race could be made. It is general impression that the disease occurred in persons of various racial and national origin, showing no predilection for any particular Stock.
12. Seasonal Incidence:- 128 cases of coronary thrombosis were admitted from Jan. 1948 to November 1951 inclusive. 81 patients were admitted in autumn and winter months while 47 in Spring and Summer months. The majority of incidence of coronary thrombosis took place in the coldest months of the year rather than in the warmest months.
13. Heredity:- five patients gave a family history of coronary thrombosis. These patients were of relatively younger age, furthermore their parents or relations had the onset or died of coronary thrombosis at a higher (older) age.

Hereditary Susceptibility appears to be more fundamental etiological factor in occurrence of coronary Sclerosis and occlusion.

14. Body Weight:- 28 per cent of patients were found to be overweight. This figure of 28 per cent for overweight patients in a Series of 128 cases with myocardial infarction is therefore not statistically significant.
15. Tobacco:- The number of smokers was rather small. 34 were heavy smokers, 17 light smokers and the rest non smokers. There is no evidence that smoking develops coronary thrombosis in this Series.
16. Alcohol:- Out of 128 patients 4 patients were heavy drinkers, 20 moderate drinkers and the rest teetotal. There is no causal relationship between alcohol drinking and coronary thrombosis in this Series. Alcohol has long been viewed with favour as a therapeutic agent in patients with coronary Sclerosis and angina pectoris.
17. Mode of onset:- In 102 patients the onset was sudden and in 26 the episode developed gradually. In 82 patients the incidence of coronary thrombosis took place during the day

and in 46 during the night. In 64% of patients the attack occurred during complete inactivity including sleep. In 12.5 per cent during mild activity and in 18.5 and 4.6 per cent the activity was moderate and vigorous respectively. The majority of attacks occurring at the non activity period were due to diminution of coronary flow and lessening of cardiac output.

The above observations indicate that physical exertion is not an essential or even important precipitating cause in most cases of coronary thrombosis, it does not exclude the possibility that it induces at least some instances of coronary thrombosis.

18. Three patients had their occlusion after an operation. The rise in blood platelets and fibrinogen after the operation, combined with surgical shock, slowing of circulation by bed rest or lessened activity attended by a diminution in blood volume and a drop in blood pressure may be attributed the possible factors associated with operation leading to coronary occlusion.
19. Angina pectoris:- Previous angina pectoris was present in 63 patients and was more in men

(53.6%) than in women (35.5%). The patients with an antecedent angina pectoris had a lower mortality (33.3%) than those without it (40%). The reason for this variation is that in angina pectoris cases the episode was gradual, beginning with an anginal pain and had time for establishing the collateral circulation while in patients without a previous angina pectoris the attack was sudden and there being hardly any time for anastomosis to establish as such the difference in mortality.

20. Pathology:- General Pathology of the disease is briefly presented.
21. Location of coronary occlusion:- 14 autopsied cases were studied. Thrombosis was found occluding the left coronary artery and its branches more frequently than right coronary artery and its branches. The occlusion was found 1-2 centimeters from the origin of these vessels. The relative freedom from occlusion of the proximal 2 centimeters of the vessels permits a free circulation through the first branch or two proximal to the occlusion by means of which a collateral circulation is maintained.

22. Location of Infarction:- In 13 of the 14 autopsied cases infarction was found in the left ventricle and the remaining one in both the left and right ventricles. This is probably due to the richer coronary anastomosis to the relatively smaller myocardial mass of the right ventricle.
23. Size of the Heart after cardiac Infarction:- Cardiac enlargement was found in 17 patients with normal or low blood pressure, of whom 10 died. Cardiac hypertrophy in the absence of hypertension was probably a compensatory reaction explained by Starling's law of the heart. It may also have been due to ischemia caused by the occlusion of coronary artery. Enlargement of heart after the myocardial infarction was a bad prognostic sign.
24. Pain:- Pain is the prominent symptom of coronary thrombosis. It was present in 117 patients. In 63 patients, it started as angina of effort and in the remaining 54 the pain was sudden and acute. In 40 cases the pain was accompanied by breathlessness and in 23 nausea and vomiting was also present, while marked restlessness was found in 21. The pain was due to myocardial ischemia.

25. Location and Radiation of Pain:- It was mostly retrosternal. There was no radiation of pain in 35 patients of whom 13 died. It radiated to left arm in 30 patients of whom 8 died, right arm in 6 patients of whom 2 died and to both the arms in 24 patients of whom 11 died. It also radiated to right shoulder 4 times, into the side of neck 15 times, through to the back 9 times, between the shoulders 4 times and into the jaws and gums 3 times. Epigasteric pain was experienced by 11 patients. Mortality was higher in the group where the pain radiated to both the arms. 11 of the 24 cases died. Higher mortality was probably due to the extensive underlying lesion (Infarction).

The findings are tabulated as follows:

Table XVII.

Showing the radiation of pain and mortality.

	No. of Cases	Deaths	Percentage
No Radiation (Retrosternal)	35	13	37
Left arm	30	8	26
Right arm	6	2	33
Both arms	24	11	46
Other Sites e.g. neck, shoulders, back etc. etc.	22	7	31

26. Silent or atypical cases of myocardial infarction:-

Pain was not experienced by 11 patients. In them there was Substernal discomfort and tightness. Dyspnoea and Shock were marked features in these cases and 6 of the 11 died. Mortality was higher in this group as compared to the pain group, i.e. the rest of the Series.

27. Premonitory Pain:- It was present in 37 cases in the series. Premonitory pain when present and recognised is of great prognostic importance as the appropriate treatment instituted at this stage may avert the impending infarction or its extension.

28. Shock:- Marked Shock was present in 38 patients and mild 52, while a feeling of exhaustion and weakness was experienced by 10 patients. A severe degree of shock was associated with higher mortality.

29. Fever was present in 71 per cent of patients. The rise of temperature was usually moderate being 99 to 101 degree fahrenheit and lasted 2-4 days in uncomplicated cases. Fever and leucocytosis appeared together in 58 cases and in 15 cases fever was without leucocytosis.

Fever and leucocytosis are probably produced by the toxic products liberated by the infarcted tissue.

30. Pericardial friction rub:- It was present in 10 patients. Although generally associated with anterior infarction it was observed in 2 cases with a posterior lesion (infarction). Its appearance renders the outlook more grave (60%) compared with (33%).
31. Blood pressure and pulse rate:- A systolic Blood pressure persistently below 100 mm. Hg was found in 25 patients of whom 16 (64%) died. Similarly the pulse rate in 36 patients was persistently over 100 beats per minute, 24 (66.6%) of these 36 died. Mortality rate thus increased when systolic blood pressure remained persistently low i.e. below 100 mm. Hg or pulse rate was persistently fast i.e. above 100 beats per minute. Majority of cases in whom death took place in the wards had both persistent low blood pressure and a fast pulse. Thus a persistent low blood pressure and a persistent rapid pulse rate have grave prognosis.

32. Leucocytosis:- A white blood cell count of 6000 to 9000 per c.mm was found in 32 cases in whom 2 died, 9000 to 12000, in 21 patients of whom 5 died, 12,000 - 15,000 in 10 patients of whom 7 died and 15,000 and above in 5 patients of whom 3 died. The mortality rate increased with the rise in white blood cell count, in fact marked leucocytosis indicated a poor immediate outlook.
33. Blood Sedimentation Rate:- Blood Sedimentation rate of 20-29 mm. per hour was found in 13 patients of whom 3 died, 30-39 mm. per hour in 12 patients of whom 5 died, 40-49 mm. per hour in 4 patients of whom 2 died and 50 mm. and above per hour in 5 patients of whom 3 died. The mortality increased with the increase in blood Sedimentation rate.
34. Other Symptoms:- These were auricular fibrillation, extra systoles, palpitation, orthopnoea, Gallop rhythm and cheyne Stokes breathing. These symptoms were not encountered frequently.
35. Congestive heart failure:- 19 patients had developed congestive heart, both left sided and combined failure in wards. 14 of these patients were males and 5 females. 11 of the

19 patients, 9 males and 2 females had died within six weeks. Congestive heart failure thus is of grave prognostic significance in patients with myocardial infarction.

36. Embolization:- 5 patients had this complication. 3 of the 5 patients died. Emboli are direct consequence of cardiac infarction. Slowing of the peripheral circulation, enforced bed rest, with lack of movement of the extremities and the associated venous Stasis are probably more important causes. Thrombo-embolic lesion in acute myocardial infarction has gained increasing significance, because of the possibility of treatment with anticoagulants and surgery. Thrombotic phenomena and emboli are of grave prognostic significance in patients with myocardial infarction.
37. Sudden death:- It is a common termination of acute myocardial infarction. 10 patients died suddenly in the wards within six weeks. 6 of these deaths were probably due to an extension of previous episode. In the remaining 4, emboli of lungs and brain were found in 3 at the autopsy and in the 4th autopsy revealed a small rupture (vent) in

the post wall of the left ventricle.

38. Treatment is discussed briefly.
39. Follow up Study:- 50 cases from 6 months to 4 years were traced. 10 of these patients had died. 4 patients died from 6 months to 1 year from the onset of coronary thrombosis, 5 died from 1 year to 1 year and 10 months and 1 died within 3 months. 3 patients^{have}/lived more than 3 years i.e. 40 months, 42 months and 47 months, and are still living.

The general health of patients as revealed by the follow up study was good in 23 cases, fair in 10 cases and poor in 7 cases.

40. From this Summary the factors influencing the prognosis of coronary thrombosis are as follows:-

A. Bad Prognosis:- (1) age, above 50 years. (2) Sex, female. (3) number of previous infarcts. (4) anterior and antero-posterior infarctions. (5) Heredity. (6) Seasons, coldest months. (7) Cardiac enlargements after the cardiac infarction. (8) Radiation of pain to both the arms. (9) Silent or painless infarction. (10) Shock.

(11) Pericardial friction rub. (12) A persistently low systolic pressure below 100 mm. Hg. (13) A persistently fast pulse rate, above 100 beats per minute. (14) Increased leucocytosis, above 12,000. (15) Increased Blood Sedimentation rate. (16) Congestive heart failure. (17) Embolization.

B. Good Prognosis:-

(1) Age, below 50. (2) Sex, male.
 (3) Initial attack. (4) Posterior infarction and the group with no electrocardiographic evidence of infarction. (5) Hypertension.
 (6) Antecedent angina pectoris. (7) Radiation of pain to left arm. (8) mild or absence of shock. (9) Temperature, normal or slightly raised for 2-4 days. (10) Blood pressure, normal or above 100 mm. Hg.
 (11) Pulse rate, normal or below 100 beats per minute. (12) White blood cell count below 9000. (13) Blood Sedimentation Rate below 15 mm. per hour.

41. Conclusion:- It is observed from this study that angina pectoris and coronary thrombosis are the different phases of the same process being caused by anoxaemia of heart muscle. The underlying factor in both the conditions

excluding the precipitating factors is the same i.e. atherosclerosis of coronary arteries. Atheromatous changes in angina pectoris however are more or less of a uniform nature, resulting usually in narrowing of the lumen of these vessels, whereas in coronary thrombosis the changes are generally of a patchy type with or without much narrowing of the caliber of coronary arteries. The precipitating factors no doubt are different, but while coronary thrombosis is caused in a person with Sclerotic coronary arteries at rest, Angina pectoris results in the same individual in excitement and with exertion.

CASE REPORTS

Case I. A male aged 59, unemployed miner, admitted on 5th September 1951, gave a history of pain in chest and shortness of breath for five days and one week respectively.

The patient was well until five days ago and was able to walk unlimited distances without pain or breathlessness.

On Thursday, 1.2.51, while out walking, he became aware of a "jagging pain" behind the lower end of his sternum, which caused him to catch his breath and stop for a minute or two until it wore off. The pain was not altered by coughing or deep breathing and did not radiate into his arms.

He had no further discomfort until Friday night, 2.2.51, when he developed a severe "gripping pain" in his chest which radiated through to his back and shoulder blades and down the outer sides of both arms to just short of the elbows. The pain was very intense and he was unable to lie in bed, being compelled to get up and walk the floor. He was not breathless but felt nauseated and sick. He suffered considerable discomfort but the pain gradually passed off, only to return with the same severity.

He has not been short of breath on exertion until recently, but he has had a short cough with yellow sputum during the winter. There has never been any swelling of his ankles. His appetite has been good and bowels regular. He has been gaining weight. He has no headaches, urinary symptoms or trouble with his eyes.

On examination the patient was found to be an obese, plethoric type of individual in no pain or discomfort. There was no cyanosis or dyspnoea. No oedema of sacrum or ankles was found. Pulse was normal, of good volume, well sustained and the vessel wall not palpable. Heart:- the apex was impalpable through very thick chest wall. The heart sounds were distinctly heard. B.P. was 150/100. Electrocardiogram revealed an anterior infarction. Blood sedimentation rate was 25 mm/hr, and white blood cells varied from 9400 to 10,000.

The patient developed pulmonary congestion and broncho-pneumonia and died on 11.3.51.

At autopsy the heart was found enlarged and hypertrophied. There was an infarct in the anterior wall of the left ventricle. Coronary arteries were atheromatous, and a thrombus was found occluding the descending branch of the left coronary artery.

Case II. A female aged 56, housewife, admitted on 20.12.48, gave a history of pain in the left leg for three weeks, and pain in right side of chest for two days.

Three weeks ago there was some bluish discolouration of inner aspect of left leg with excruciating pain, sufficiently severe to keep her awake at night. Was treated with Kaolin poultices and injection of morphia to relieve pain.

Two weeks ago an area of reddish discolouration appeared from the medial femoral condyle to the groin, which was extremely tender to touch, though not painful; it felt very hot to touch. Some swelling of the lower leg appeared about the same time. Therapeutic régime was continued until the redness disappeared and she felt very much better for about two days.

Two days ago the patient rose from her bed and after a few minutes was suddenly seized with a sharp pain in the right side of the chest. There was no cough or spit and she did not become breathless. The pain was made worse by cough or deep breathing. The patient came out in a cold sweat with alternate sensations of heat and cold. She felt rather feverish. There was no previous history of shivering attacks or haemoptysis.

No dysuria or frequency. Urine slightly reddish on day of admission. Good control of bladder and bowel. No stress incontinence. No cough, spit, or nasal discharge. She becomes rather breathless on normal exertion and the ankles swell when she stands for any length of time. She has been off her food since the onset of illness. She has had a "tired heart" with attacks of dizziness while standing in queues.

On examination the heart was found to be normal in mid-clavicular line. Pulse rate was 100, and the pulse was regular; the wall was not palpable. Blood pressure was 110/70, and white blood count 7,400. She died on 31.12.48.

At autopsy the myocardium of the entire left ventricle and that of the anterior third of the right ventricle and anterior half of the interventricular septum was soft, friable and congested. The appearance was that of very recent infarction. No thrombus was found in any of the larger branches of the coronary arteries and the main trunks and ostia were of normal calibre. These vessels displayed only inconspicuous atheromatous involvement.

Case III. A female aged 65 admitted on 12.11.51, gave a history of vomiting, diarrhoea for 24 hours, and pain in the epigastrium for 8 hours.

The patient is a known diabetic. She rose yesterday (Sunday) morning and gave herself the accustomed dose of 20 units of Globin Insulin, had breakfast and did her housework.

Early in the afternoon after lunch, she passed a loose stool. There was no blood in it and it was not painful to pass.

She noted that she had a vague pain across the lower chest at the level of xiphi—sternum. It did not radiate and was neither strangling and gripping in character nor related to breathing and coughing. At 9 p.m. she had a meal of canned salmon sandwiches. She passed another loose stool before retiring. The pain still remained in her chest, gnawing in character and present all the time, made now worst by laughing. She did not feel feverish and fell asleep.

At 2 a.m. she awakened, coughed a great deal but had no spit, and then vomited a whitish fluid containing food, neither green nor brown in colour. She was repeatedly sick till the morning and also passed some more loose stools.

The pain in the chest was still present and she now felt short of breath.

No headache. Eyesight good. Appetite poor for two weeks. Bowels regular. No pain or difficulty in passing urine. No frequency. Two weeks ago she had a cold in the nose and a slight reproductive cough with fever.

E.C.G. revealed a posterior infarct. Blood pressure was 90/50. Blood sedimentation rate 12 mm/hr and white blood count 124,000.

The patient died on 18.12.51.

At autopsy myocardial infarction involving the posterior wall of the left ventricle and posterior part of the interventricular septum was found. Occlusion of circumflex artery 1 cm. distal to its origin from the main trunk of the left coronary artery was also found. The coronary vessels revealed atheromatous changes.

Case IV. A male aged 53, warehouseman, admitted on 19.5.51, gave a history of severe substernal pain for the last 3 days.

The patient was perfectly well until three days ago, when, while awaiting a tramcar, he developed a

severe substernal pain radiating down both arms on the inner side and lasting two to three minutes, after which he felt quite normal. That evening in bed he had a second sharp attack lasting only a few minutes.

On 20.5.51, at 2 a.m. he awoke with a severe pain and tightness of the chest, the pain radiating to the hands. He was restless, pale but did not feel sick and there was no vomiting. During the course of the day he has had three further attacks.

Appetite normal up to three days ago. Bowels regular. No abdominal pain or flatulence. Micturition normal. He has been a very healthy man.

W.B.C., 10,200. E.S.R. 4 mm/hr. Electrocardiogram was taken and showed evidence of myocardial damage.

He was treated by rest and anti-coagulants and although he continued to have a tingling sensation in both hands and arms for some fourteen days after admission, he settled down and made an uneventful recovery. He was discharged on 23.6.51.

Case V. A male aged 60, boiler stoker, admitted on 1.10.51, gave a history of intermittent pain in the chest for the last two months.

This patient was quite well until two months ago when he experienced a fairly severe pain over the sternum and epigastrium. The pain was dull "as if something was sticking". He was in bed at the time; the pain lasted a few minutes and passed off completely. He had several similar attacks following this during the next eight weeks, each time lasting a few minutes. Sometimes these came on during exertion, sometimes while he was excited, but he always kept quite still and they passed off. Two days before admission, while going into his garden, he experienced another attack, which did not quite disappear. The pain worsened the next day, and by that evening was very severe, requiring an injection to allay it.

The pain continued the next morning and he began to complain of nausea, vomiting clear fluid twice in small quantity.

Appetite poor, bowels regular. No dysuria. Slight reproductive cough. No swelling of ankles. No breathlessness.

This man has always been well apart from a long standing complaint of weakness of the legs and unsteadiness while walking. This began

insidiously twelve years ago when he began to notice fatigue after walking and a tendency to fall to the left at night. This has slowly become worse, especially in the last few months.

On examination the patient was found distressed with pain, with good colour and no disorientation. Pulse 60 per minute with occasional extrasystoles. Blood pressure 160/100. The heart sounds were muffled and closed over all areas. No oedema of ankles. No E.C.G. was taken.

The patient was given 10,000 units of Heparin intravenously six-hourly followed by 1.2 gm. of Tromexan daily.

During the first night the patient collapsed and became almost pulseless, pulse remaining irregular following this. Cheyne Stokes Breathing was treated by Coramine and oxygen and morphine sedation. Patient died at 5 p.m. on 2.10.51.

Case VI. A male aged 48, butcher, admitted on 9.7.51, gave a history of substernal pain of three days' duration.

This patient retired in good health on Thursday evening. At 1 a.m. on Friday morning (3 days ago) he experienced sudden acute substernal pain radiating down inside of left arm as far as the left wrist and upwards into the jaw and side of neck. Shortly afterwards he vomited. The pain is described as an "acute stabbing ache" which gradually eased towards morning but he got no sleep, tossing about and being unable to find any position which eased it. He was again sick at about 3 a.m. With the help of several sips of brandy he attempted to go to his work as usual on Friday morning, but shortly after commencing in the shop the pain came on worse than ever. He returned home to bed, and his doctor was called that evening. He was given an injection of Heroin but had a disturbed night with further chest pain.

On doing heavy work he has never experienced any chest pains. However, eight weeks ago, again whilst in bed, he experienced a similar attack of substernal pain. There was no sickness and no radiation of pain to jaw. A slight attack of pain occurred five months ago. Appetite good. Weight steady. Micturition normal but has had bouts of pain while passing urine some years ago.

On examination the patient was neither shocked nor restless but was rather pale and anxious.

Apex beat was seen in the 5th interspace in the midclavicular line. There was no thrill. Pulse rate

was 75 per min. The pulse was regular, of good volume, well sustained and the vessel wall was not palpable. The heart sounds were closed in all areas. Blood pressure was 110/65.

The blood sedimentation rate was 3 mm/hr and white blood cells 9,200.

The patient has a text book history of coronary thrombosis. He was immediately put on anti-coagulants. Electrocardiogram was taken one week after admission. All the twelve leads were negative. At this stage there had been no change in the cardiac rhythm. Blood pressure has remained constant (although low - 110/70) and there had been no further chest pains.

The white blood cells had remained constant at 9,200 and the blood sedimentation rate the same as on admission, i.e. 3 mm/hr. Anti-coagulant therapy was stopped.

On 31.7.51 a straight X-ray of chest and barium meal was done. No lesion was found in stomach, oesophagus or duodenum. A degree of left ventricular enlargement and a degree of prominence and unfolding of the aortic arch was seen in the chest film. He was allowed up on the 18th. A check-up E.C.G. was done on the 25th of July, i.e. eight days after the first one and sixteen days after admission. This electrocardiogram was also negative, all twelve leads within normal limits. He was kept under observation for one month in the ward and there has been no repetition of symptoms. He was discharged on 4.8.51.

Case VII. A male, aged 56, agent, admitted on 29.5.51, gave a history of pain in the chest, radiating into left arm and hand, accompanied by breathlessness.

Two days ago at 6.30 p.m. this man suddenly experienced severe substernal pain which was crushing in nature. At the time of onset he was sitting at tea. The pain radiated down the inside of his left arm and hand.

He was unable to remain still and walked the room and eventually went to bed, where he tossed and turned till his own doctor arrived and gave him an injection. Next morning when he awoke the pain had gone and he felt well. He remained at rest in bed and felt well till he was awakened this morning with the same severe crushing substernal pain, radiating into the left arm. He was restless, sweating and felt sick. He was given gr. 1/4 of morphine by mouth.

On examination the patient did not appear shocked. He was lying still and his colour was good.

He complained of substernal pain. He had coronary thrombosis three and a half years ago but no other serious illnesses.

Pulse 90 per min., regular, fair volume. The apex beat was in the 5th space in midclavicular line. The heart sounds were of tic tac quality.

He was given morphia on admission, i.e. 11 a.m. At 2 p.m. the patient suddenly collapsed, his colour became ashen and he was sweating profusely. He died at 2.5 p.m. No E.C.G. was taken.

This patient having had coronary thrombosis three and a half years before, had been in good health till two days before admission, and despite sedation and complete rest in bed, he died as a result of further occlusion or rupture of the ventricular wall.

Case VIII. A male, aged 67, admitted on 9.7.51, gave a history of breathlessness and substernal pain.

This patient went to his doctor this evening complaining of breathlessness. He was being examined, stripped on a couch, having his chest percussed, when he experienced a sudden acute substernal pain radiating upwards to his neck and jaw. He described the pain as stabbing and almost unbearable. Afterwards he felt very restless and was not comfortable either lying down or standing up. He did not feel nauseated and did not vomit. There was no history of pain radiating down the arms but he gave a vague history of pain at the tip of the right shoulder and pain radiating through to the back.

He also complained of cough, worse at night, when he was on day shift. He brought up white frothy sputum, never blood stained. These symptoms, with the breathlessness have become progressively worse in the last four to five months. He frequently had attacks of nocturnal dyspnoea. There was no swelling of the ankles.

Five months ago he experienced a severe attack of substernal pain associated with vigorous exercise, e.g. firing a boiler. He has had several attacks of chest pain of lesser degree associated with exercise such as walking up a hill.

Bowels regular; micturition normal, 2-3 times at night; no day frequency or dysuria.

This man has always been healthy.

On examination the patient was found cyanosed, sweating, breathless, his blood pressure was 90/65 and pulse imperceptible.

He was given morphine and intravenous Aminophylline. The patient died at 6 a.m. the next day.

Case IX. A male, aged 65, night watchman, admitted on 12.8.51, gave a history of pain starting from the left hand and into the chest, slight cough with sputum.

The patient got up at 11 a.m. and wanted to go for a wash when he experienced a very sharp "crushing pain" starting in the left hand and radiating upwards into the chest. The pain was severe enough to double him up. He was covered with profuse sweating and the injection which his doctor gave him did not relieve much of the pain. He did not have undue shortness of breath along with the pain. The pain was at first paroxysmal in character, waxing and waning in intensity, but since an hour had become continuous.

Eleven years ago he developed a cough and sputum. He was investigated fully and was said to be suffering from chronic bronchitis and hypertension.

Eight weeks ago he developed a cold which became worse and he called his doctor, who gave him some medicine which relieved him. On and off, since this period he has had pain in the arm and chest which used to come off independent of any exertion. The pain was eased by "brown tablets" given by his doctor. The pain used to last at least half an hour and again tend to reappear.

He has attacks of cough with sputum, which was blackish to yellow in colour. He never had any blood in the sputum. He became short of breath when he stooped or exerted himself. No oedema of the feet.

Eleven years ago he had two "black-outs" but never had any fits. He used to suffer from headaches but was free now.

He did not sleep well and had to be given sedation. Bowels ^{day} regular. No retention. Micturition three times during, twice at night.

Electrocardiographic examination on admission showed an antero-posterior myocardial infarction.

He was treated with a course of Heparin and Tromexan, followed by graduated physiotherapy and discharged on 6.10.51.

Case X. A male, aged 49, fishmonger, admitted on 4.5.51, gave a history of severe pain in centre of chest of twelve hours' duration.

Yesterday afternoon this man took part in an observer post exercise and while carrying a heavy piece of machinery he experienced a tight pain in the middle of his chest and was short of breath. On putting down the weight the pain disappeared immediately and he completed the exercise, but on returning home he felt "done in", and went to bed.

This morning he felt well, and went to work but about lunchtime when walking up a hill he felt the pain return. He struggled home, went to bed and was given an injection which eased the pain.

He came downstairs this evening and while sitting felt the pain suddenly, which was extremely severe, substernal, vice-like in nature and radiating down the inner side of the left arm to the elbow. He rolled round the floor in agony and was described by his daughter, a nurse, as being first cyanosed, then ashen, breathless and sweating. He vomited twice. The pain continued at the same pitch for some two hours, during which time he continued to roll about the floor, and only eased when he was given gr. 1/4 of morphia.

For some months now he has had tight "wind-like" pains in his lower chest, usually after meals, and these he attributed to certain foods not agreeing with him.

On examination the patient was found extremely ill. His blood pressure was 110/65. He was cyanosed and examination of his cardiovascular system revealed a regular feeble pulse, rate 90, and an enlarged heart, apex beat being in the 6th space in the anterior axillary line. Triple rhythm was audible over the base of the sternum, and two days after admission a loud generalised to and fro pericardial friction rub was present.

During the first week he needed almost continuous sedation by morphine for his precordial pain, and was also given oxygen therapy more or less continuously.

He was given anticoagulants in doses according to clotting and prothrombin time and during his second week in hospital he improved dramatically.

During his third week he felt and looked very weak and on the afternoon of the 21st day he suddenly collapsed and died almost instantaneously.

It was presumed that death was due to further extension of the thrombosis or pulmonary infarction.

Case XI. A female aged 58, housewife, admitted on 19.3.51, gave a history of tightness behind the top of the sternum.

While playing bridge last night she was seized by a tightness in her chest behind the upper part of her sternum. This did not radiate but she felt faint although she did not become unconscious. She broke out in a cold sweat. She was a little short of breath. This tightness is still present but is not so severe as it was.

Since Christmas she has been wheezing during the night. No cough. No swelling of the ankles. Her feet have always been cold. Her fingers become white and numb for some time.

Appetite good. Bowels regular. Has to get up at 4 a.m. to pass urine. No headaches. Sees a halo round the electric light.

On examination the patient was found pale, clammy, slightly dyspnoeic and not in great pain. There was no cyanosis, clubbing of fingers, oedema or venous distension.

Pulse 64 per min., regular in time and force, good volume and well sustained. Vessel wall was palpable but not sclerotic. Blood pressure 180/120.

Apex beat was in the 5th interspace on mid-clavicular line. There was no thrill and the apex beat was forceful. Heart sounds were loud and pure in all areas. Blood sedimentation rate was 25 mm/hr. Electrocardiogram revealed an extensive recent anterior infarct.

The patient was treated with Heparin, thereafter with Tromexan 1.29 gm daily reduced seven days after admission to 0.6 gm daily to maintain prothrombin time at 30.36 seconds.

During this period, i.e. from 19.3.51 to 5.4.51, no complications occurred and her heart sounds improved in quality with a gradual rise in blood pressure from 110/80 to 140/85.

On the morning of April 5th, at 7.50 a.m., she was seen collapsed and after a few breaths, expired. She had not previously complained of pain in the chest or a choking feeling.

Possible causes of death:- (a) Pulmonary embolus, or (b) Ruptured heart.

Case XII. A male, aged 67, retired miner, admitted on 15.4.51, gave a history of pain in the chest and breathlessness for the last three years.

This man was perfectly well till three years ago, when he began to be aware of a gripping pain in his chest which came on as he walked to and from his work. This pain was always behind the breast bone, gave a feeling of tightness, and his jaws, gums and side of neck were always affected at the same time. During the attack he felt breathless and almost seemed to choke. The pain was relieved almost immediately by rest and he was then able to carry on. He continued to work but one month before retiring age, was forced to give up his work on account of the frequency of the attacks of pain.

In the last two years he has had a lot of flatulence after meals, with pain behind the breast bone. The pain is eased after the wind is brought up.

He also noticed that on walking he got a cramp-like pain in the calf of the right leg, which was relieved by rest.

More recently in the past nine months, his relatives have noticed a change in his colour, and he himself has noticed that his feet have been cold and he has been troubled by "pins and needles in his legs". During the last month he has been more breathless and felt tired.

The tongue has been a little sore recently. Bowels constipated for the last three weeks. He experienced difficulty in starting micturition.

On examination the patient was found lying quietly in bed. No cyanosis, jaundice or oedema.

Pulse 120 per min., regular in time and force, good volume, wave normal and vessel wall palpable but not tortuous. Blood pressure 160/110. Heart:- apex beat in the 5th intercostal space inside the midclavicular line. Heart sounds were tic tac in quality.

Electrocardiogram revealed right ventricular hypertrophy and right bundle branch block.

This patient had generalised atheromatous change as shown by his anginal attacks and claudication. X-ray showed no real evidence of ventricular enlargement but anterior and posterior tibial arteries showed marked calcification. His irregular pulse and tachycardia while in the ward were attributed clinically to a coronary occlusion, but E.C.G. failed to confirm this. Digitalisation was therefore begun and with a fall in pulse rate the patient began to feel better.

The patient died suddenly on 5.5.51. His sudden death must be attributed to coronary occlusion.

Case XIII. A male, aged 64, forrester, admitted on 22.1.51, gave a history of tightness in the chest and severe breathlessness of a few hours' duration.

The patient was well until three years ago when he was confined to bed for six weeks. His illness at this time developed with a sudden pain between his shoulder blades and he was forced to go home to his bed. The pain was sharp in character and did not radiate into chest or arms immediately, but he did experience a mild pain down the inside of his left arm several days later while in his bed. He was a little breathless at the time and was told that his heart was affected but he believed he had "gastric flu".

After he recovered from this he experienced occasional "twinges" of pain, which started in the back and moved through to the front when he walked through ploughed fields or did a little more strenuous work than he was accustomed to. He has also had occasional twinges of pain in the inner aspect of his left arm similar to the pain described above, which only came on while he was working during the day and have never been experienced at rest.

This afternoon, while hammering holes in a wall, he suddenly experienced a very unpleasant feeling of tightness in the chest and at the same time he broke out into a "hot sweat". For a moment he had the conviction that he was about to die. Then he recovered slightly, dropped his hammer and managed to move to a seat. The tightness continued and he noticed that he was having great difficulty in breathing and also that his respiratory rate was increased. The sweating continued, and since the tightness did not pass off, he allowed his foreman to take him home in a lorry. He went to bed, still with the tightness and breathlessness and was perspiring profusely. He took a little spirits and water, and noted considerable improvement in the tight feeling. A little later he was given an injection of morphia which relieved all his symptoms.

Appetite good. Bowels constipated. No trouble with micturition.

On examination the patient was found to be a plethoric, middle aged man in no pain or discomfort but slightly breathless at rest in bed. He was not cyanosed.

Pulse rapid, 100 per minute, regular in time and force. Good volume, well sustained. Vessel wall palpable but not tortuous. Blood pressure 138/74.

Heart:- apex beat not visible through thick chest wall but palpable in the 6th interspace 4 inches from

the midline. The first sound at the apex was almost replaced by a loud long blowing systolic murmur which was propagated into the axilla but the second sound was closed and of good quality. The sounds at the base were obscured by the breath sounds but the aortic second sound is loud and there were no murmurs present.

The blood sedimentation rate was 16 mm/hr. and white blood cell count 15,000.

E.C.G. revealed a posterior infarct.

This patient was treated with anti-coagulants and made steady and uninterrupted progress, and was discharged on 4.3.52 symptom-free.

Case XIV. A male, aged 75, retired miner, admitted on 16.1.50, gave a history of precordial pain of seven days' duration.

The patient felt quite well until seven days ago, when a sharp aching pain came in front of his chest. The pain did not involve his neck or arms. He was given some tablets to help him and relieve the pain. The patient has remained in bed ever since. The patient had no history of angina or of headaches or of intermittent claudication. He does not wake up at night feeling breathless and did not get breathless when climbing a hill.

He has to get up two or three times at night to pass urine. He has a good appetite generally, but it has not been good during the past weeks. His bowels move regularly.

Seven years ago he had abscesses in the chest following an accident in the pit, and it was operated upon.

He drinks four or five pints of beer daily. Nothing particular in the family history.

On examination the patient was found to be an elderly man, plethoric, rather heavy and fat and somewhat deaf.

Pulse 72 per min., regular in rate, rhythm and force. Vessel wall impalpable. Blood pressure was 112/80.

Apex beat in the 5th interspace in the mid-clavicular line. Heart sounds:- faint in all areas.

The blood sedimentation rate was 27 mm/hr and white blood count 7,000.

E.C.G. revealed an antero-septal infarct.

The patient was kept at rest in bed and after three weeks he had a course of physiotherapy prior to getting up. He was discharged on 18.2.50.

Case XV. A male, aged 74, retired miner, admitted on 24.11.49, gave a history of intermittent precordial pain of three weeks' duration.

This patient was quite well until three weeks ago. While he was in bed at night he woke up with a severe crushing pain in his chest, in both arms and the left side of the neck. He writhed about in bed and felt as though he was choking. The following day the pain had considerably diminished and he went to his work. But that night the pain occurred again and the patient was unable to go out to his work next day. He saw his doctor, who advised him to go to bed and he has remained in bed ever since.

He has had frontal headaches for some time and has also been breathless, particularly when going uphill. He wakes up at night feeling breathless about midnight and has to sit up in bed to try to get his breath. Often he has to get up and go to the window before he can get his breath again. These attacks have been occurring frequently for seven or eight years. He has never had any swelling of his legs. He has never had any precordial pain like this.

He has always been healthy and there is nothing suggestive of heart disease in the family history.

He smokes four ounces of tobacco a week and a glass of beer occasionally.

On examination pulse rate 78 per min., regular in rate and rhythm. Blood pressure 116/60.

Apex beat was found in the 5th left interspace five inches from the midline. Heart sounds:- in mitral area first sound was accentuated. At the lower end of the sternum third heart sound was audible. In all other areas heart sounds were normal. Slight breathlessness was present while lying flat in bed.

Blood sedimentation rate was 25 mm/hr and the white blood count 16,000.

E.C.G. was not taken.

The patient was diagnosed as a case of coronary thrombosis. He was immediately started on a course of Heparin and kept at rest. Some degree of left-sided cardiac failure developed and was accompanied by hypostatic pneumonia. This was controlled by a

course of sulphonamides. Shortly after the Heparin was stopped, a right-sided pulmonary infarct occurred, signs and symptoms of which disappeared after a week and the patient appeared to be making a good recovery. Ten days later another sudden coronary thrombosis occurred and he died instantaneously.

Case XVI. A male, aged 59, school janitor, admitted on 14.11.49, gave a history of severe precordial pain of 24 hours' duration.

This patient was operated on for a carcinoma of the rectum on 25.10.49 and was discharged to a convalescent home on 25.10.49. There he has been getting up and has been having his colostomy dressed. He has been on daily baths and dressings to his perineal wound.

Last night, 13.11.49, he developed a severe precordial pain over his lower sternum. He was sweating, sick and very restless. The pain was partly relieved by alkalis, but recurred and increased in severity and it was found necessary to give him morphine. He slept for the rest of the night and the pain was much less severe in the morning. His blood pressure previously was 220/118, last night it was 190/100, and this morning had fallen to 130/80.

Before admission to hospital he had noticed that on walking more than a quarter of a mile he began to have pains in the back of the calf muscles of his legs. There was, however, no history of breathlessness or of angina pectoris although some years ago he had slight gripping pain in his chest which lasted for a few hours and then disappeared.

In 1919 he had pneumonia and made a good recovery. In September 1949, he was operated on for carcinoma of the rectum and discharged to convalescent home where the present symptoms developed.

His one sister has died of angina pectoris. In the rest of the family there was no history of heart disease.

On examination the pulse was 80 per minute, regular in time and force. Vessel wall thickened and blood pressure 125/90.

E.C.G. revealed a posterior infarction. The blood sedimentation rate was 32 mm/hr and white blood count 13,000.

This patient developed a coronary thrombosis while convalescing from a colostomy following removal of rectal carcinoma. He was kept at rest

and made an uneventful recovery.

He was re-admitted on 2.4.50 with a sudden severe pain over the lower end of the sternum and died on 10.4.50.

At autopsy an old and a new infarction in the posterior wall of the left ventricle were found. The heart was hypertrophied, weighing 500 grms.

Coronary arteries showed severe atheroma and the anterior interventricular artery 1 cm. from its origin was blocked by pale grey ante-mortem thrombus.

Case XVII. A female, aged 61, housewife, admitted on 18.11.50, gave a history of precordial pain.

This patient has had angina of effort for the past three years. This afternoon, 18.11.50, she was seized by a raw burning precordial pain which radiated down the left arm. It lasted all afternoon and that evening she became very breathless and was given 1/4 gr. morphia. This relieved the pain.

She was breathless on exertion. Appetite good. No cough. No swelling of ankles. Bowels regular. No urinary symptoms. No central nervous system symptoms.

Her father had died of myocardial degeneration.

On examination she was found dyspnoeic and in pain, sitting up in bed.

Pulse 100 per min., regular in time and force, good volume, well sustained. Vessel wall palpable and a little thickened. Blood pressure 140/100. Apex beat was in 5th interspace in mid-clavicular line. There was a triple rhythm at mitral area. The sounds were closed in all areas.

Blood sedimentation rate 14 mm/hr. and white blood count 13,000.

Electrocardiogram revealed an anterior septal infarct extending to the posterior.

It was a case of coronary thrombosis in a woman who previously had angina of effort. A good recovery followed Heparin and Tromexan therapy.

Case XVIII. A male, aged 51, park house officer, admitted on 8.1.51, gave a history of severe pain in the chest of three hours' duration.

This morning, 8.1.51, while walking to his work, the patient was struck by a severe pain in his chest. It came on very suddenly and was extremely acute and he felt that he was "going to choke" with the fear of impending death. The pain mounted in intensity and spread across his chest and down into the right side of his abdomen, but did not radiate to his arm or neck.

When the pain came on at first he was able to walk 200 yards to his hut, but the pain then became unbearable and he was unable to sit down, being forced to walk about by the intensity of the pain. He felt nauseated and became very pale, breaking out in a cold sweat. He was taken home in a car and at that time felt "all in". Once in bed he felt sick and vomited a small quantity of food and a few spots of blood. He was given an injection of morphine by his doctor but the pain remained until he was admitted to hospital.

For the last two weeks he has complained of a "sharp gnawing pain" in his left shoulder and arm which he thought was due to neuritis. The pain was present day and night and was not related to exercise but was relieved by tablets which he took every four hours. It has remained unaltered since his attack of retro-sternal pain today.

For the past two or three months the patient has been short of breath on exertion but has had no nocturnal dyspnoea. He has no swelling of the ankles. He has had gastric flatulence for some time but no nausea or vomiting. His appetite was good and the bowels regular. He thinks he has been gaining weight recently.

There is no history of heart disease in his family. He smokes 20 cigarettes a day and drinks beer and spirits.

On examination the patient was found stout, middle-aged man, slightly dyspnoeic and in pain. He was slightly cyanosed.

Pulse 70 per min., regular in time and force, pressure poor, vessel wall not palpable. Blood pressure was 110/64.

Heart:- apex beat was neither visible nor palpable and the heart sounds were indistinct in all areas with marked splitting of the first sound in the mitral area. No murmurs or friction rub were present and there was no oedema of sacrum or ankles.

E.C.G. revealed a posterior infarction involving the apex.

This patient was treated with anticoagulant therapy. The infarct was a posterior one involving the apex and associated initially with heart block but this disappeared before discharge, when the patient was very fit with a good exercise tolerance. His weight was reduced by two stones six pounds.

Case XIX. A male, aged 52, laundry manager, admitted on 12.2.51, gave a history of pain in chest and left arm for one day.

This patient has always been a nervous individual and has had a lot of worry. In May 1950 he was in hospital with a duodenal ulcer and nervous debility, but from the time of his discharge until 11.2.51 he has felt very fit and although he has had numerous business troubles he has always been able to keep "on top of them".

On the day before admission, 10.2.51, after having breakfast in bed, the patient felt faint and dizzy and perspired freely, feeling that he was "going to faint". He had no pain at this time and recovered in approximately ten minutes, but two hours later, while still in bed, he had a very similar attack and kept fighting against the feeling that he was "going to pass out". After the initial sweating sickness and breathlessness had passed off, he became aware of a "searing pain" behind the upper part of his sternum and dull numbness in his left arm from his shoulder to his finger tips. The pain in his chest lasted for four hours with decreasing intensity but did not radiate into his back or neck. He did not vomit at the time but was sick at 2 a.m. this morning. He has had intermittent fainting attacks for the past three years, these being precipitated by small things, e.g. the sight of blood or by someone cutting their finger, but he has never experienced any pain in his chest or arms and has never injured himself during an attack, nor has he passed urine or faeces. There has been no history of fits.

The patient has been breathless on exertion for the past five years and this comes on when he attempts to hurry or climb steps. He has had palpitations but never any swelling of his ankles.

Appetite good. No pain associated with meals. Bowels tend to be constipated. No urinary symptoms.

His father died of heart disease and his grandmother had V.D.H. He smokes 20 cigarettes a day and drinks very little alcohol.

On examination the patient was found to be thin-faced, middle-aged man with an anxious look on his face. Not distressed but rather nervous.

He has no cyanosis or oedema. There was no dyspnoea and the neck veins were not distended.

Pulse 44 per min., regular in time and force, poor volume, not well sustained. Vessel wall not palpable.

Heart:- apex beat neither visible nor palpable. The heart sounds were faint and indistinct in all areas but there were no murmurs present and no pericardial friction rub.

Blood sedimentation rate was 6 mm/hr and the white blood count 7,500.

Electro-cardiogram revealed a posterior infarct.

The patient was treated with anti-coagulants. He made a good recovery and although still nervous, left the hospital on 24.3.51 feeling well.

Case XX. A male, aged 69, silver finisher, admitted on 12.2.51, gave a history of shortness of breath on exertion, pain in chest and both arms for two years and very severe retrosternal pain for a few hours.

The patient was well until two years ago when he became breathless on exertion, this being precipitated by climbing a small incline and going up steps. One year later he began to experience a sharp pain in his chest and back on exercise, and he was breathless with these attacks but the pain lasted for approximately 15 minutes and passed off when he rested. Recently the pain has become more severe and has radiated into both arms and between the shoulder blades and is precipitated by walking up the smallest incline although he can walk a good distance on the flat without pain.

This morning after climbing the steps into a tramcar, the patient suddenly developed "a very severe tearing pain" across his chest which radiated through to his back and he felt as if he was "encased in a jacket of pain". He felt short of breath and had a choking sensation but did not know whether to lie down or stand up to get relief and eventually fainted. He cannot remember what happened to him during the next seven minutes but when he came round he was free of pain but had a dull frontal headache. He did not vomit.

He did not have frequent headaches but his vision has been blurred for the past year and he wears spectacles.

For the past seven years he has suffered from severe cramp-like pains in his thighs and calf muscles.

which come on mostly at night in bed. These pains have never come on with walking but his feet have always been cold in bed.

Appetite good. Weight has been increasing. Bowels tend to be constipated and he has seen bright blood on the outside of his stools when he has been extremely constipated, but he has never passed black tarry stools.

He has had frequent epistaxis recently. He passes a large amount of urine four or five times during the day and twice through the night but it has never been discoloured.

On examination the patient was found to be a stout middle-aged man with good colour, in no pain or discomfort, and looking younger than his years.

He has no cyanosis, dyspnoea or distension of neck veins. No abnormal pulsation in the neck and no oedema of the sacrum or the ankles.

Pulse regular in time and force. Good volume. Vessel wall palpable and slightly tortuous.

Heart:- apex beat not visible but palpable in the 5th interspace just outside the mid-clavicular line. B.S.R. 15 mm/hr. W.B.C. 6,000.

All over the precordium there was heard a very harsh systolic murmur which almost completely replaced the first sound and appeared to originate in the aortic area. At this area the systolic murmur was propagated into the neck and the second sound was inaudible but a soft blowing diastolic murmur replacing the second sound was also heard down the left margin of the sternum.

Electrocardiogram showed no evidence of coronary occlusion but revealed left ventricular hypertrophy.

This patient clinically was a case of coronary thrombosis but the E.C.G. suggested left ventricular hypertrophy, no definite evidence of an infarct, but did not exclude the presence of myocardial damage. The patient did very well with a course of anti-coagulant therapy and left the hospital on 24.3.51 feeling well and entirely free of symptoms. He was advised not to return to work for at least one month.

Case XXI. A female, aged 47, housewife, admitted on 19.2.51, gave a history of retrosternal pain for five days.

The patient was perfectly well until she woke on 15.2.51 with constricting tearing pain behind the sternum. She did her normal day's work on that day but the pain gradually became worse. At first it was

intermittent but on 16.2.51 at night, and on the 17th and 18th February, 1951, it remained constant. The pain radiated up her neck across both sides of her chest and into both arms and passed down the inner side being worse on the left. The pain was accompanied by a choking sensation, extreme restlessness and dyspnoea. She also felt cold and shivery.

She was not normally breathless and although she has had not true nocturnal dyspnoea, she sometimes wakes up at night and thinks she has forgotten to breathe. She has always had a slight cough with clear sputum. She occasionally has slight swelling of her ankles.

Appetite good. Bowels regular with aperients. No urinary symptoms. No headaches. No signs of effort.

On examination the patient was found pale, shocked and in severe pain and dyspnoeic.

Pulse rate 90 per min., regular in time and force, good volume, well sustained. Vessel wall not palpable. Blood pressure 140/80.

Heart:- apex beat in 5th space within the mid-clavicular line. Heart sounds of equal intensity closed in all areas. No pericardial friction rub.

Electrocardiogram revealed posterior infarct extending to the apical region.

The patient was treated with anticoagulants, Aminophylline and phenobarbitone and made an excellent recovery. She was discharged on 31.3.51 symptom free.

Case XXII. A female, aged 67, housewife, admitted on 25.2.51, gave a history of severe pain in chest of a few hours' duration.

For over a week this patient had a pain in her chest in the precordial region at night, associated with dyspnoea. She obtained relief by sitting up. The pain was not acute but was gripping in character. This pain was not constantly present in the precordium; sometimes it was felt between the shoulder blades and sometimes in the wrists. On the night of the 24th she was awakened by an acute pain, again gripping in character all over the chest and between the shoulders, which radiated into the arms. She was not dyspnoeic. She has had this acute pain continuously but has gained some relief from physeptom intramuscularly.

Appetite good. Bowels regular. No urinary symptoms. No swelling of the ankles. Occasionally

she has slight non-productive cough. She has had pain and tightness in her chest on exertion, which is relieved by rest. This has not troubled her since she was in hospital one month ago.

The patient had two similar attacks five years ago and one month ago.

There is no history of heart disease in the family.

On examination the patient was found in good colour. There was no dyspnoea or pain and she was able to lie flat in bed.

There was no oedema, cyanosis, dyspnoea or clubbing of the fingers.

Pulse rate 74 per min., regular in time and force. Vessel wall palpable but not sclerotic. Blood pressure 100/70.

Apex beat not palpable or visible. Heart sounds were faint but pure, first and second sounds almost equal in intensity. No thrills, murmurs or pericardial friction rub.

No E.C.G. was taken.

A case of coronary thrombosis in a woman of 67 who had two previous attacks. Anti-coagulant therapy was commenced but she died 8½ hours after admission.

Case XXIII. A male aged 60, retired, admitted on 31.12.50, gave a history of severe pain in the chest for two hours.

This patient was in a state of collapse and unable to give a good history but he gives evidence of having been in hospital in Sydney, Australia, with "coronary occlusion" and "myocardial degeneration" in 1945 while working there during the war.

Since 1945 the patient has had angina of effort, his last attack being three weeks ago. He has also been breathless after undue effort for several years and on the day of admission was breathless and experienced a slight pain in his chest after hurrying up a slight incline to the bus from his caravan in a frostbound field. The pain soon passed off and he was able to carry on until this evening, when he was seized by a very severe "gripping" retro-sternal pain which radiated into both arms as far as the finger tips and over his forehead. It also passed through to his back and he "collapsed in the street" where he lay for half an hour before admission. He did not vomit but felt nauseated and broke out in a cold sweat.

He has had no swelling of ankles or palpitations. He has been breathless on exertion for several years. No nocturnal dyspnoea. Bowels regular. No urinary symptoms.

There is no history of heart disease in the family. He drinks and smokes occasionally.

On examination he was found to be a grey-haired middle-aged man, going bald, lying in extreme pain and cyanosed. There was no clubbing of the fingers or venous distension.

Pulse was 70 per min., regular in time and force, poor volume and not well sustained. Vessel wall not palpable. Blood pressure 130/72.

Heart:- apex beat not visible or palpable. The heart sounds were very faint and of poor quality in all areas and tic tac. There was no oedema of sacrum or ankles.

E.C.G. revealed a recent posterior infarct with probably also an old anterior infarct.

Admitted with a history of coronary thrombosis in 1945 and angina of effort with breathlessness on exertion. This patient on admission proved to have had a second coronary thrombosis with posterior infarct. He was treated with anticoagulants till he expressed his desire to leave hospital against medical advice. He did not do this however when the time came and remained in bed for another three weeks until the time of his discharge when he had a reasonably good exercise tolerance but was not entirely free of pain in his right arm.

Since discharged on 3.2.51 this man has remained fairly well, but some three weeks ago he began to be aware that his breathing was laboured. This has continued and two nights ago he suddenly became acutely breathless and thought he was about to die. This attack settled fairly rapidly but last night he again became acutely breathless. He attempted to go to his work but was unable to get there and had to call from the street for help. He was given an injection and his breathing has been less laboured since.

For the last three weeks his mouth has been very dry and he has been drinking a large amount of fluids.

This patient was re-admitted on 15.6.51 in left ventricular failure. He was given intravenous Aminophylline and Digoxin. He was dehydrated and there was acetone in his breath and as such he was put on a saline drip for twelve hours. In five days his respiratory rate had returned to normal. He was up on 30.6.51 and discharged on 7.7.51 on Digoxin 0.25 mgm. T.I.D.

Case XXIV. A male, aged 60, engineer, admitted on 24.1.51, gave a history of severe sternal pain of 24 hours' duration.

The patient was well until two years ago when he developed a very severe "cramp-like pain" in his chest on several occasions when he was doing heavy work and climbing ladders. When the pain came on he felt as if something was "being torn out of him", but it only lasted for a few minutes and passed off when he rested. It did not radiate into his arms or neck and did not pass through his back. He was not breathless.

Twelve months ago the attacks of pain became more frequent and lasted for a longer period of time, being precipitated by decreasing amounts of exercise. It then came on after climbing slight inclines and after rushing for trains but never while he was at rest.

In April of May of last year he remembers having one particularly severe attack of pain which persisted for 25 minutes and gradually wore off although he kept walking all the time.

At 10 a.m. on the morning of the day before admission, while at his work, the patient was suddenly gripped by an extremely severe tearing "cramp-like" feeling in his chest which radiated into his left arm and was most severe at the medial side of his elbow. It did not pass into his jaw or through to his neck but he felt dizzy, light-headed and very weak, and he broke out in a "profuse hot sweat". He lay down immediately and noticed that he was a "terrible grey colour".

He was taken home and put to bed, but was unable to find a position in which he could get relief from the pain. His doctor gave him a bottle but this did not help the pain and he vomited a few hours later. This appeared to relieve the pain but he had a dull sensation in his chest and arm throughout the night.

The patient has complained of flatulence and waterbrash for several years but has had no abdominal pain related to meals, or vomiting.

He has been breathless on exertion recently and he thinks that his ankles may have been slightly swollen, especially the left at the site of an old injury. He has no cough or spit.

No urinary symptoms. Appetite very good. Weight has been increasing.

There is no history of heart disease in the family. He smokes moderately but does not drink.

On examination the patient was found to be a middle-aged white haired man with good colour and slight tinge of cyanosis about the lips and ears. He was not in pain or discomfort.

He had no dyspnoea, no swelling of ankles or oedema of sacrum. Neck veins were not distended.

Pulse 60, regular in time and force, good volume and well sustained. Vessel wall palpable but not tortuous.

Blood pressure 124/92. Heart sounds in all areas were indistinct. No murmurs or friction rub was heard.

E.S.R. 25 mm/hr and white blood count was 11,200.

Electrocardiogram revealed a posterior infarction.

A case of coronary thrombosis in a man who had a previous history of angina of effort for two years, was treated with anticoagulant therapy and made an uninterrupted recovery. He was discharged on 4.3.51.

Case XXV. A male, aged 63, retired miner, admitted on 20.11.50, gave a history of a severe retrosternal pain for five hours.

At 4 p.m. this afternoon after a quiet day in bed at home, the patient developed a very severe "gripping pain" in the precordium, which moved across into his right breast and then into both shoulders and down both arms to the finger tips. The pain was very intense and caused him to roll about in bed, unable to find a position to give him relief. He sweated considerably but did not vomit and the pain remained intense until 7 p.m., when he received an injection and the pain became less severe but did not go away. He was breathless during the attack.

The patient was in hospital in May 1948 with a coronary thrombosis and since then has had frequent retrosternal pain on exertion which passed off in a few minutes if he rested and was also relieved by some "special pills". He has also had retrosternal discomfort at rest but no real pain until today.

For the past six months he has been breathless on exertion, but never at rest and there has been no paroxysmal dyspnoea. He has never had any swelling of the ankles.

Appetite poor. He feels that large meals bring on the pain. He has been gaining weight recently. There is no history of cardiac disease in the family.

On examination the patient was found to be a middle-aged man with high colour. He was not cyanosed or breathless and was not in pain. There was no cyanosis or dyspnoea.

Pulse 80 per min., regular in time and force, vessel wall not palpable. Blood pressure 156/86.

Heart:- apex beat not visible but palpable over an area of the size of a half-crown, in the 6th interspace in the midclavicular line. Heart sounds were of poor quality especially at the mitral area where there was a marked splitting in the first sound and a reduction of quality in the second. There were no murmurs or pericardial friction rub. No oedema of ankles or sacrum.

The blood sedimentation rate was 15 mm/hr and the white blood cell count 9500.

Electrocardiogram revealed an old posterior infarct and a recent anterior infarct.

This patient had a posterior infarct in 1948 and was readmitted with a very recent anterior infarction on 20.11.50. His progress was slow and for a time there was evidence of left ventricular failure, but eventually he made a good recovery and was discharged with a reasonable exercise tolerance and free from pain and breathlessness. While in hospital he was found to have sugar in his urine but a blood sugar curve showed this to be of low renal threshold type.

Case XXVI. A male, aged 60, painter, admitted on 16.7.50, gave a history of pain in the chest for two weeks.

For the past two years he has been attending his doctor because of dizzy turns and feeling light-headed. He was unable to lie on his left side at night because of the dizziness. He does not fall down when he has these dizzy attacks and does not vomit.

Two weeks ago he began to experience pains in the chest, which were not related to exercises but which were more severe when the cold wind blew on his chest. The pains would sometimes come on even when he was in bed or sitting in a chair. The pains began behind the lower part of the sternum and passed up into both shoulders and down the outer aspect of the arms as far as the wrists. He also felt the pain down the front of the right leg and sometimes in the calf of the left leg. He said that he could touch the tender spot on the sternum and below the clavicles. The pain was at times so severe that he could not get rest and he had to keep moving from one position to another.

He was never breathless and there was no swelling of the ankles. He also had severe temporal headaches which were relieved by aspirin phenacelin tablets.

Appetite good. Bowels tended to be constipated. Weight was increasing.

He had been a healthy man. There was no history of cardiac disease in the family.

On examination he was found to be a middle-aged man who complained of chest pain. He was well built and highly coloured. There was no cyanosis and no finger clubbing.

Pulse 65 per min., full, regular in time and force. Vessel wall not palpable.

Blood pressure was 110/65.

The blood sedimentation rate was 15 mm/hr and the white blood count 7000.

Electrocardiogram revealed posterior infarction. X-ray showed enlarged left ventricle.

This patient, who has had a history of angina pectoris for which he was treated with tab. glyceryl trinitrite gr. 1/130, developed coronary thrombosis at the age of 60. He was treated with anticoagulants and rest as usual and made an uneventful recovery. He was discharged on 27.8.50.

Case XXVII. A male, aged 63, engine driver, admitted on 23.10.50, was found collapsed at work at 2.45 p.m. on the day of admission.

This patient was in good health until a month ago when he had an attack of sickness during the night and stayed off his work the following day. He did not know the cause of his sickness and he had no pain with it. He was able to go to his work the next day, but complained of a dull "empty feeling" in his stomach for the following fortnight. He was never sick but felt nauseated.

On 10.10.50 he was at his work and developed a "sick feeling" which came on at 12 a.m. and lasted until he got home at 4 p.m. He then had a meal and developed a very severe "gripping pain" behind his sternum, which passed through to his back and into both shoulders, especially the left, and finally down the inside of right arm to his little finger. He was unable to remember how long the pain lasted but said that it was severe for three hours and then became less intense. He went to bed immediately it came on and was able to lie still but awakened with

a dull retrosternal pain the next morning. He went to work the following day and had no further attacks of pain until today.

This afternoon he signed on at 1.15 p.m. and waited for his engine until 1.50 p.m. when he mounted the footplate. There he developed a sharp pain between his shoulder blades, which radiated up the back of his head. He dismounted and walked 200 yards after this, still with the pain and then it seemed to become very severe and choking in character, moved to the front and spread into both arms and he collapsed. He could not remember anything until he was admitted.

He has had harsh short cough for the past four weeks. There was no breathlessness or swelling of the ankles. Bowels regular and no urinary symptoms.

There was no family history of cardiac disease. The patient works two shifts and has had irregular meal hours for years. He was a moderate smoker and took alcohol occasionally.

The patient was a heavy muscular man, plethoric type, in a state of "collapse", conscious and free of pain but pulseless, grey and covered in a cold sweat. There was no loss of power in arms or legs or cranial nerve lesion.

He was not breathless and there was no clubbing of the fingers or swelling of the ankles.

Pulse poor volume, not well sustained. Vessel wall just palpable. Blood pressure 94/62. Heart:- apex beat was not visible through the thick chest. Heart sounds were very faint and of poor quality in all areas. No murmurs or friction rub were heard.

The blood sedimentation rate was 35 mm/hr. Electrocardiogram revealed a recent posterior infarct with possibility of infarction also of the front of the heart.

This patient was admitted in a severe degree of shock from coronary thrombosis. There was no previous history of anginal pain and he had another attack the day after admission, when he was on Heparin. He was mentally confused from the first day and this became progressively worse, as did his urinary and faecal incontinence. His heart sounds were very weak and he developed a pericardial rub six days after admission. E.C.G. showed evidence of a recent posterior infarct and probably an anterior one. He died on 13.11.50.

At autopsy a massive infarction of the posterior wall of the left ventricle was found with a haemopericardium and possibly a small rupture of the posterior wall. There was old adherent pericarditis.

The heart was hypertrophied and weighed 56 gms. There was atheroma of the aorta.

Coronary arteries showed marked calcification with patchy narrowing of their lumenia. The left coronary artery as it ran in the atrioventricular groove contained possibly a very recent antemortem thrombus of small extent at a point where the lumen was much narrowed. Infarction of kidney and spleen was also found. There was a small cerebellar infarct.

Case XXVIII. A female, aged 55, housewife admitted on 16.10.50, gave a history of retrosternal pain of 24 hours' duration.

On 15.10.51 the patient had had three severe spasms of pain behind the sternum. This pain radiated through to the back between her shoulder blades, and down her right arm. She had a similar attack two days ago when she was drying frocks. Her right hand and arm went numb and she dropped the frocks. She did not lose consciousness but felt "squeamish".

Her appetite was good. Bowels constipated and no urinary trouble.

She was very dyspnoeic on exertion and has not been breathless at night. She has had no swelling of her ankles. She has never had a haemoptysis. She had no trouble with headaches.

She has always had a tender appendix but never operated upon. Six years ago she was said to have a gastric ulcer.

Her mother, sister and brother all died of angina. The sister had an attack three months ago. She smoked about 15 cigarettes daily but did not drink.

The patient was a florid, cyanosed woman in pain.

Pulse fast, regular in time and force. Fairly good volume. Vessel wall palpable and sclerotic. Blood pressure 140/84.

Heart:- apex beat was in the 6th interspace, within midclavicular line. Heart sounds were closed and pure in all areas. No pericardial rub or murmurs.

Discs showed arterio-sclerotic changes.

Blood sedimentation rate was 18 mm/hr. Electrocardiogram showed an anterior infarction.

The patient was treated with anticoagulants and rest, but her condition deteriorated and she died on 31.10.50.

At autopsy an infarction of the lateral wall of left ventricle was found.

Heart was of average size, weighing 380 gms. Extending from the apex to a point 2 cm. distant from the superior limit of the left ventricle the myocardium was infarcted. At the apex the infarct was mainly posterior. Midway up the left ventricle the infarct extended round the lateral wall.

Coronary arteries:- showed throughout considerable irregular atheromatous calcification and thickening. The circumflex branch of the right coronary artery was completely occluded by firm adherent antemortem thrombus. The left coronary, though considerably atheromatous contained no thrombus in either its main portion or in any of its branches.

Case XXIX. A female, aged 60, housewife, admitted on 24.7.50 with a history of pain in chest for several hours.

The patient was unconscious and the history was obtained from a friend and doctor's letter.

For two years she had not been well and has suffered from "heart attacks" and tiredness. Her doctor said that she was known to have been hypertensive and had a pain in her chest last night, which was still there this morning and she also had pain in her wrists. He found her blood pressure 90/80 and crepitations at the bases of her lungs. She was coughing up a frothy sputum. He had given her Morphine gr. 1/4 and hyoscine 1/100 at 9 a.m. and also 10,000 units of Heparin.

When taken to the ambulance at 9.30 a.m. she was able to talk but by the time of admission one hour later she was unconscious and could not be roused.

On examination she was found to be a well covered woman, lying unconscious with quiet breathing and normal temperature.

Pulse rate 110 just palpable in neck. Heart sounds were inaudible. Crepitations were found at both bases of lungs but no consolidation. Liver, spleen and kidneys were not enlarged. All the limbs were flaccid and the deep and superficial reflexes absent.

Discs normal, apart from arterio-sclerotic changes. Blood pressure could not be obtained.

A case of coronary thrombosis in a hypertensive subject who died from left ventricular failure after some initial improvement from her state on admission.

In spite of digoxin, heparin and luminol she died at 3 a.m. on 26.7.50.

Case XXX. A male, aged 48, foreman engineer, admitted on 11.7.50 gave a history of praecordial pain of 4 hours' duration.

On the evening before admission to hospital the patient felt slight twinges of pain on the left side of the chest while reading his paper, but he did not think much about it and went to bed. During the night he was awakened by a severe pain across the chest on both sides of the sternum and extending down both arms as far as the elbows. The patient described the pain as burning in character and worse than anything he had experienced before. It made him come out in a cold sweat and shiver. He called his doctor who gave him I.M. morphine, grs. 1/4 et atropine gr. 1/50.

He had been treated for gastric ulcer for the last six years, and was discharged from the Forces because of this ailment.

There was no history of cardiac disease in the family.

Examination:- A middle-aged man not in any great distress, but speaking with difficulty, and dyspnoeic.

There was no cyanosis, but some dyspnoea was found on talking.

Pulse 60, full, regular in time and force. Vessel wall not palpable. Blood pressure 90/60.

Heart:- apex beat was in 4th interspace $1\frac{1}{2}$ " outside the midclavicular line. Heart sounds were closed but at the mitral area there was a blowing systolic murmur.

Blood sedimentation rate was 15 mm/hr.

Electrocardiogram showed posterior infarction.

A case of coronary thrombosis in a man aged 48, who was treated with I.V. Heparin, 10,000 units 6 hourly. He made a satisfactory recovery and was discharged home on 26.8.50.

Case XXXI. A female, aged 69, housewife, admitted on 10.7.50, with a history of pain in the chest and left arm of four hours' duration.

At 8.30 p.m. on 10.7.50. after a normally active day, she was out walking when she was seized by a severe pain in the front of the chest over the middle of the sternum, which radiated to the left arm at the shoulder and was felt from there along the lateral aspect of the arm to the middle of the forearm. Her husband helped her home walking very slowly for a quarter of a mile. When she got home she felt sick, and sat down, and when she tried to get up to make some supper, she felt dizzy and had to sit down again. The pain was about as severe from onset to admission 3½ hours later.

About a year ago she was off her food and feeling tired, and she went to her doctor who gave her tablets for bloodlessness and told her her heart was slightly weak. She picked up quite quickly however and seemed to have been quite well since.

Appetite fair but tends to be constipated and takes cascara every second night. Always passed water once at night since an operation for prolapsed uterus 11 years ago. She has had diabetes since the operation which has been controlled adequately by diet. Her doctor stated that her systolic blood pressure was usually over 200 but has dropped to 160/100 after the pain.

There is no history of cardiac disease in the family.

Examination:- the patient was pale, slightly cyanosed, not in pain following morphine on admission.

Pulse 68, regular in time and force. Vessel wall palpable. Apex beat was in the 5th interspace in midclavicular line. Heart sounds were closed. There was no oedema.

Blood pressure 158/100.

White blood cell 7600.

E.C.G. revealed anterior septal infarction.

Blood cholesterol 242 mgm%.

This old lady, who was a mild diabetic, was admitted with a history suggestive of coronary thrombosis. E.C.G. confirmed the myocardial damage but not any gross infarction. She made a good recovery and now feels very well. Her blood pressure was 100/94 at the last reading, this being lower than what her doctor had known it to be before admission.

Case XXXII. A male, aged 59, stableman, admitted on 25.12.50, gave a history of pain in the chest and breathlessness of two weeks' duration.

This patient was well until two weeks ago, when one night while walking to get his evening paper, he suddenly developed a very severe "gripping" pain behind the lower end of his sternum, which made him "catch his breath" and stand still for ten minutes until it passed off a little. The pain did not radiate to his neck or arms and did not go through his back. He was a little breathless at the time and this has become progressively worse during the past two weeks. At first the dyspnoea was on exercise alone but it forced him to give up his work two weeks ago and in the past three days he has been compelled to sit up in a chair all night because of breathlessness.

He has had "a few" attacks of pain in his lower chest and upper abdomen in the past two weeks, and these came on exertion.

Eight days ago he developed a short productive cough and noticed that the sputum was bloodstained. This has continued intermittently ever since and today his spit has been very thick and red. He has not had any swelling of the ankles, or palpitations. No headaches or eye disturbances. Appetite good until a few days ago. Bowels regular and no urinary symptoms.

He was a smoker and took alcohol a little. There was no history of cardiac disease in the family.

On examination the patient was found to be a thick set heavy elderly man, cyanosed and very breathless, coughing up blood stained sputum.

There was clubbing of the fingers, and the neck veins were distended but there was no visible arterial pulsation.

Pulse rapid, irregular in time and force, poor volume, not well sustained. Vessel wall not palpable. Heart:- apex beat not visible or palpable through thick chest wall. Heart sounds of poor quality in all areas. No murmurs or friction rub present. There was slight oedema of ankles and sacrum.

Blood sedimentation rate 15 mm/hr and the white blood count 7000.

E.C.G. revealed auricular fibrillation, - ventricular rate 140 per minute. Recent myocardial infarction involving apex and extending to posterior surface. Appearance of right ventricular hypertrophy in VI, probably associated with the infarction.

Admitted with a history of severe gripping retrosternal pain and evidence of heart failure with auricular fibrillation. Electrocardiogram revealed an apical infarct and the patient responded well to treatment with Aminophylline 0.2 G. T.I.D. and Digoxin. He was discharged on 2.2.51, having been reduced in weight with a good exercise tolerance, normal sinus rhythm and electrocardiographic evidence of a healing apical infarct. He was asked to continue with Digoxin 0.25 mgm T.I.D. for five days per week and also instructed to find a less strenuous job.

Case XXXIII. A male, aged 72, admitted on 18.12.50, with a history of pain in the left chest anteriorly of one day's duration.

He was perfectly well till yesterday morning when he awoke feeling cold and sweating. He also felt nauseated but did not vomit and was able to get up and go out for his morning papers. He had no pain while out walking, but once he returned to the house and sat down he developed a sharp pain to the left of the lower end of the sternum, which lasted approximately an hour and forced him to go to bed. The pain was not very severe and did not cause him to turn about in agony, but it was associated with a numbness of the radial side of the left arm, and both sides of the jaw. It was aggravated by coughing and deep breathing, but only lasted for a few seconds. He had no history of anginal pain but has been slightly breathless on climbing stairs. There was no cough or swelling of the ankles. Appetite good. Bowel regular. No urinary symptoms. No headaches. He has had nothing like this before.

A year ago his legs gave way under him and he fell in the street. His doctor told him that it was due to "blood pressure".

There was no history of heart disease in the family.

On examination the patient was found to be of good colour, slightly breathless. There was no cyanosis, venous distension or oedema.

Pulse 110, regular in time and force. Good volume, well sustained. Vessel wall palpable but not tortuous. Blood pressure 130/90. Heart sounds faint and almost obscured in mitral area by a marked pericardial friction rub. Sounds were closed and pure in all areas.

Blood sedimentation rate 42 mm/hr. and white blood cell count was 12500. Electrocardiogram revealed a recent antero-septal infarction.

Admitted on 18.12.50 with a history of retro-sternal pain and breathlessness on exertion, the patient was in a severe degree of shock and had pericardial friction rub, which lasted for only two hours. He made poor progress and developed paroxysmal fibrillation with signs of congestive failure which responded to Digoxin and Quinidine. On admission the patient's electrocardiogram showed the presence of a recent antero-septal infarct and when repeated on 2.1.51, there appeared to have been extension. The patient died on 5.1.51.

Autopsy revealed cardiac infarction with abscess formation and pericarditis. The anterior half of the left ventricular septum and the whole of the apex was the site of a recent infarct.

The heart was hypertrophied, weighing 560 Gms. Coronary arteries were atheromatous in patches with occasional calcification but no thrombus could be found.

Abdominal aorta showed moderate atheroma.

Case XXXIV. A male, aged 81, retired estate manager, admitted on 10.4.50, gave a history of chest pain of four years' duration.

For about four years this patient has had pain on exertion, at first when digging in his garden, and later when walking up a hill. This pain was a gripping pain over the lower end of the sternum and in the upper gastrum. It passed off if he stood still but sometimes a warm drink is required. He also said that frequently after such an attack he was unable to do anything but rest for the remainder of the day. His pain also appears to come on about two hours after food and he was unable to say what relieved it.

Apparently on 7.4.50 he was not feeling well and had some praecordial pain in the afternoon, which became much worse and since then has been persistent in the same place, i.e. upper gastrum and lower third of the sternum. It was severe and gripping in nature and appears to be there all the time though several periods of more severe pain have occurred lasting about an hour at a time. He was very restless during those spells and threw himself about in bed and was given morphine to control the pain.

Since 7.4.50 he has been off his food but has not been sick. His bowels have not moved but he has passed flatus. He tends to be constipated and may miss several days.

He has always had a good appetite and tends to overeat. No bladder trouble.

For some years he has had fibrositis with pain in the neck, shoulder and arms. There was no history of cardiac disease in the family.

He was a heavy pipe smoker and took beer occasionally.

Examination - A very red faced, heavily built man with spidery veins all over his face. He talked rather irrelevently but was not disorientated. He complained of pain, constant in the lower third of the sternum and upper gasterium.

Pulse 88, regular in time and force but of moderately poor volume. Vessel wall palpable. Blood pressure 170/110.

Heart:- apex beat in 5th interspace just outside midclavicular line. All sounds weak and the first sound split, best heard just medial to the apex.

White blood cell count was 12,000. Electro-cardiogram revealed an anterior myocardial infarction.

The patient was an old man with a four years' history of anginal pain terminating in a large anterior myocardial infarct, difficult to be diagnosed clinically but clearly demonstrated by electrocardiogram. He gradually deteriorated during his stay in the ward and died seventeen days after admission.

Case XXXV. A female, aged 69, housewife, admitted on 25.10.48, gave a history of pain in front of her chest, tight and burning in character, of six weeks' duration.

About 14 weeks ago the patient became aware that when walking fast or going up stairs, she became more easily breathless than formerly. Later this breathlessness was accompanied by a burning substernal discomfort and a tightness in the chest. This did not radiate in any direction and was not relieved by stopping or standing still, although she did notice that when she continued walking the pain gradually became more severe. This complaint was not accompanied by any abdominal distension or gastric flatulence. She was treated fairly successfully with some form of analgesic by her own doctor.

On the night of 24.10.48, while lying in bed, the same pain as before suddenly seized her, but on this occasion it radiated into both arms and down to the middle finger. She felt nauseated and vomited several times and broke out into a cold sweat. In a vain attempt to obtain relief she moved from side to

side in her bed and got up and sat by the fire.

Her appetite was poor for the past week. Bowels regular and no urinary disturbances. She suffered from occasional headache. There was loss of sensation in the limbs.

There was no history of heart disease in the family.

On examination the patient was found lying in bed, not breathless or in severe pain. There was no cyanosis or dyspnoea. No oedema of the ankles.

Pulse 65, regular in time and force, good volume. Vessel wall palpable.

Blood pressure 135/78.

Heart:- apex beat in 5th place inside mid-clavicular line. The heart sounds were faint and closed at all areas. There was no pericardial friction rub heard.

Electrocardiogram revealed a posterior infarct.

This patient was treated with rest and routine conventional treatment and made a good recovery. E.C.G. findings have shown uninterrupted improvement.

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